The American Journal of Surgery

EDITED BY THURSTON SCOTT WELTON, M.D.

January 1944

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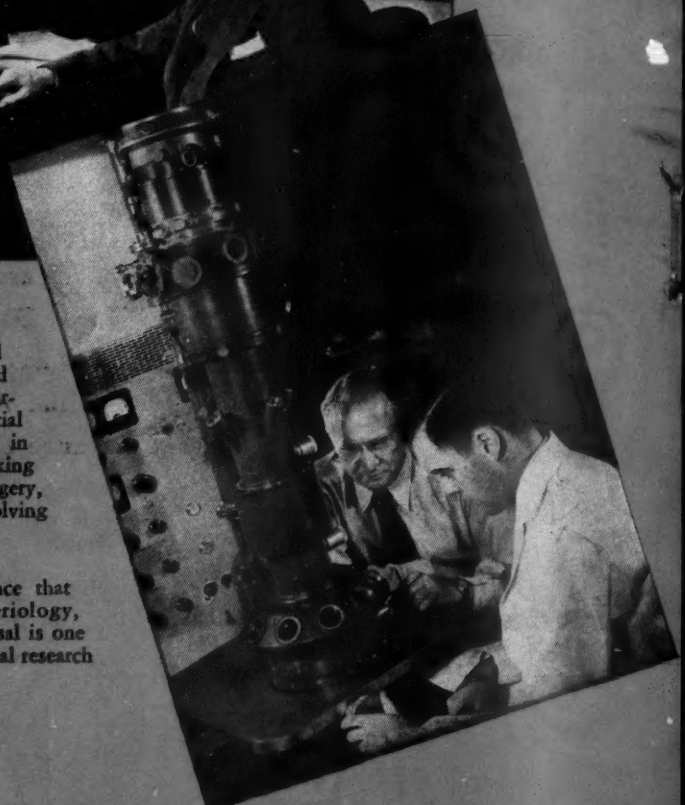

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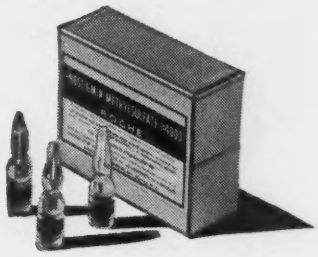

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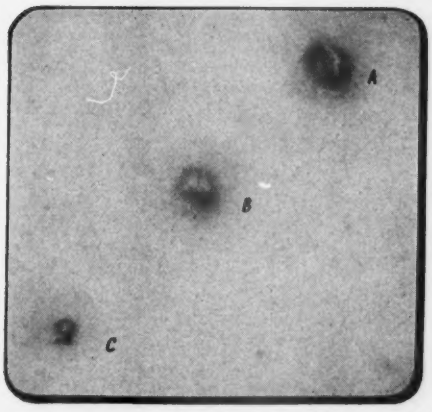

Human skin reaction at 6 days to 0.05cc. of tubing fluids injected intradermally. Ulcer has developed at site A (brand A), initially characterized by intense reaction. Early moderate reaction at B (former Curity) has subsided with no tissue necrosis. Initial slight irritation at C (new Curity) has almost disappeared.

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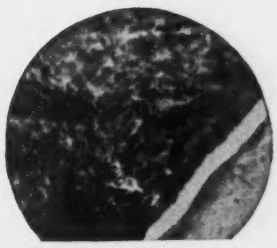
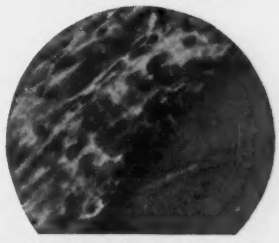


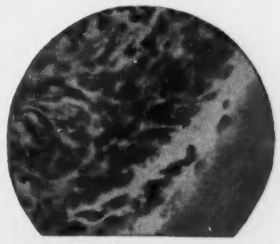
This tissue reaction is intense. A closely packed layer of leucocytes extends back some distance from the suture.

FORMER CURITY



General reaction of considerably less intensity. Moderate leucocytic response. Sub-adjacent to suture are macrophages and fibroblasts in appreciable numbers.

NEW CURITY



Predominating here are macrophages and fibroblasts with very few leucocytes present, which indicates minimal tissue reaction after 7 days.

[1] *"Surgical Gut (Catgut) Tubing Fluid as a Tissue Irritant," "Hydrocarbon Content of Non-boilable Surgical Gut Tubing Fluids," "Irritant Properties of Tubing Fluids as a Factor in the Tissue Reactions Observed with Surgical Gut (Catgut)." Ann. Surg., 118:269, 285, 288.*

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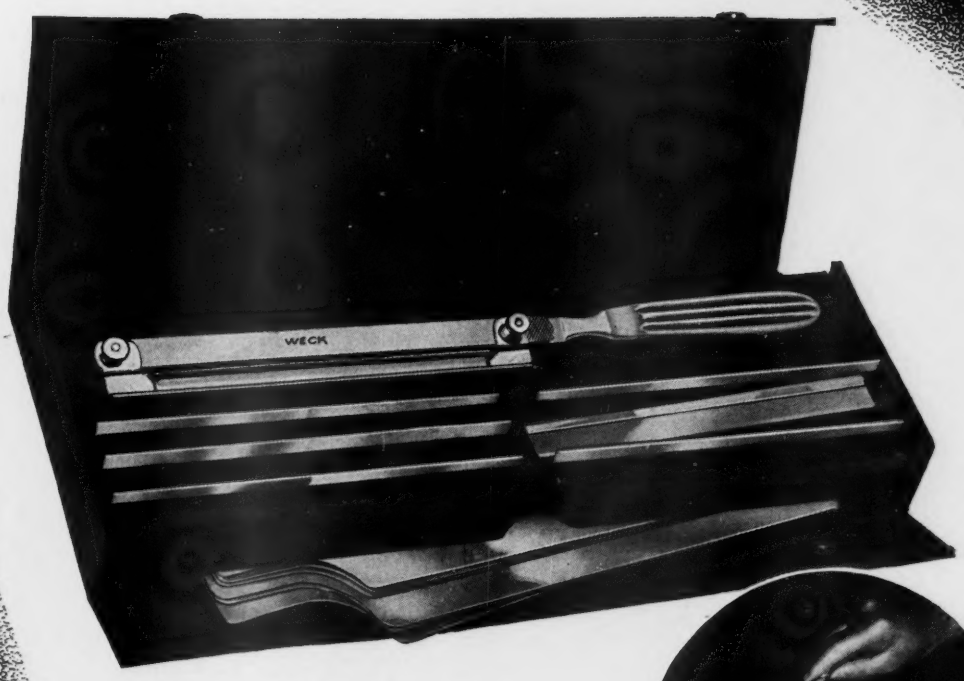

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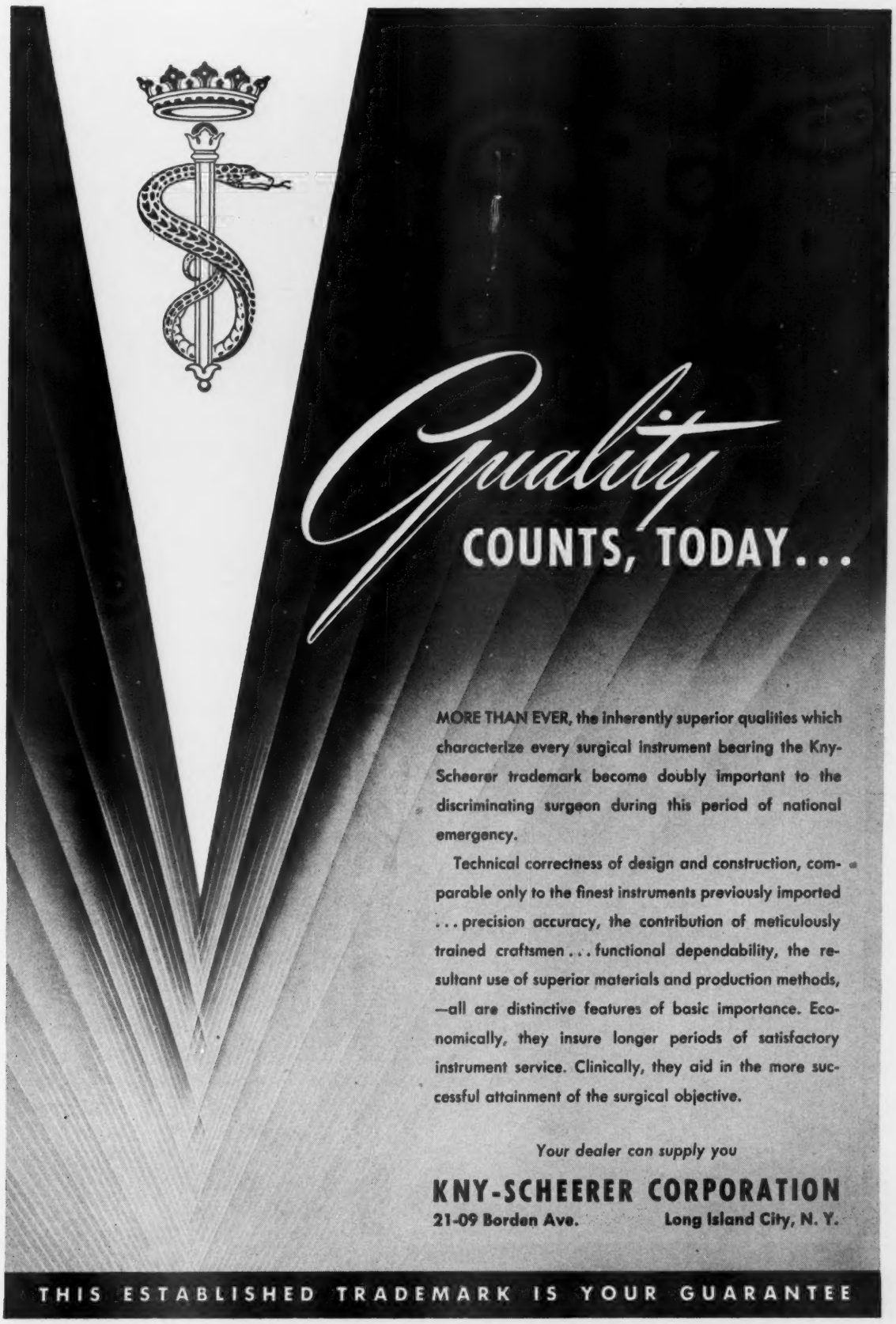
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Australian and New Zealand J. Surgery, 12:119, Oct., 1942

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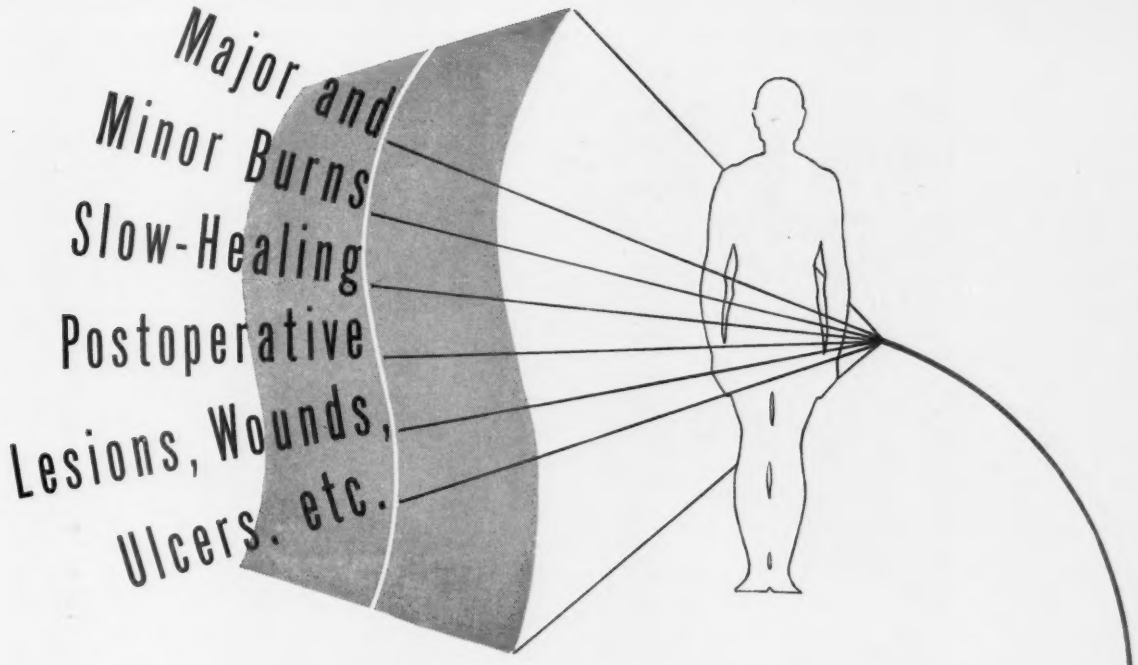

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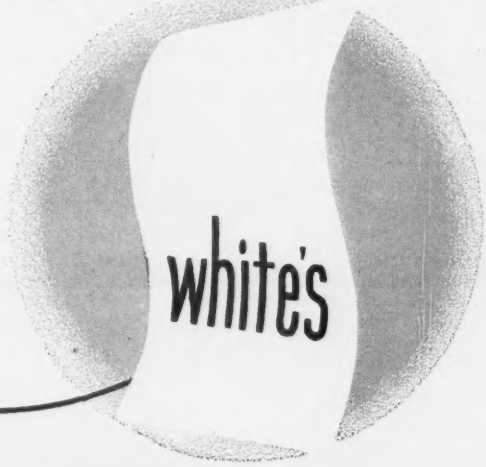

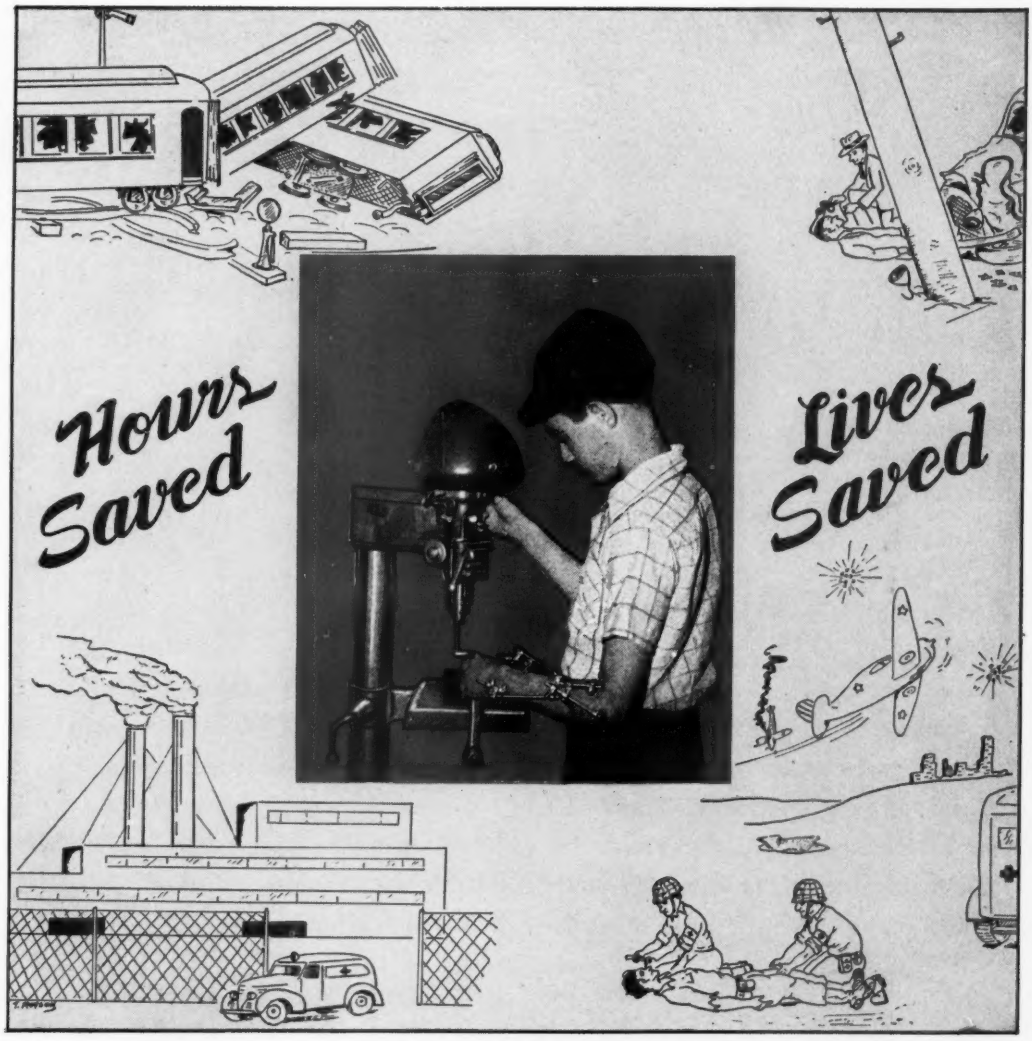

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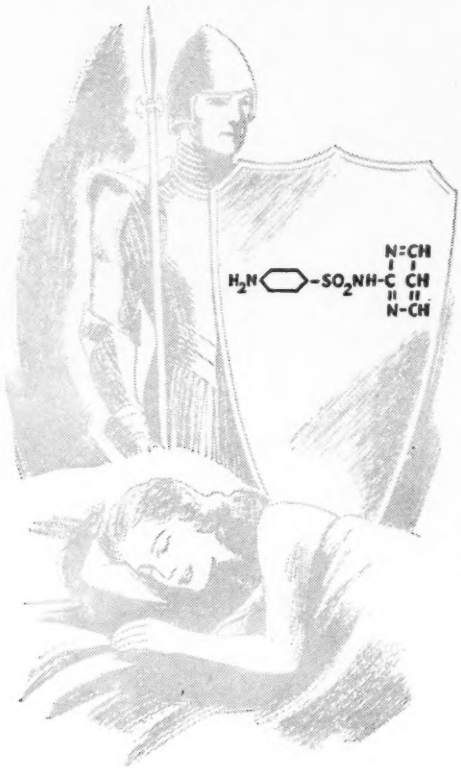

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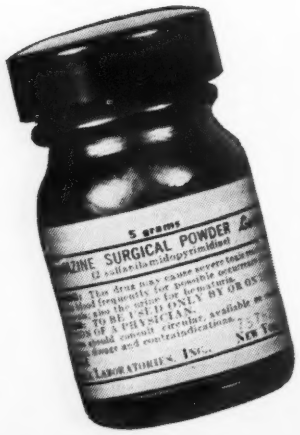

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*Johnson, R. E., et al.—Journal of Nutrition, Dec. 1942

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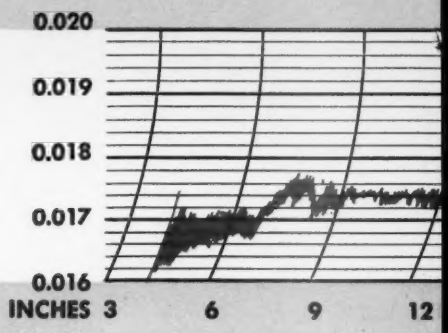

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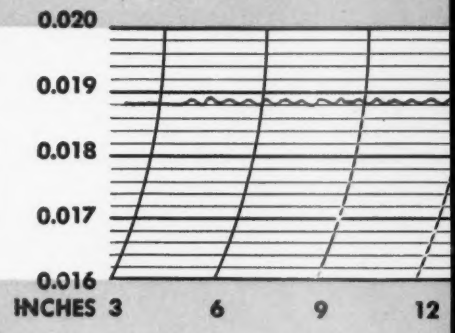

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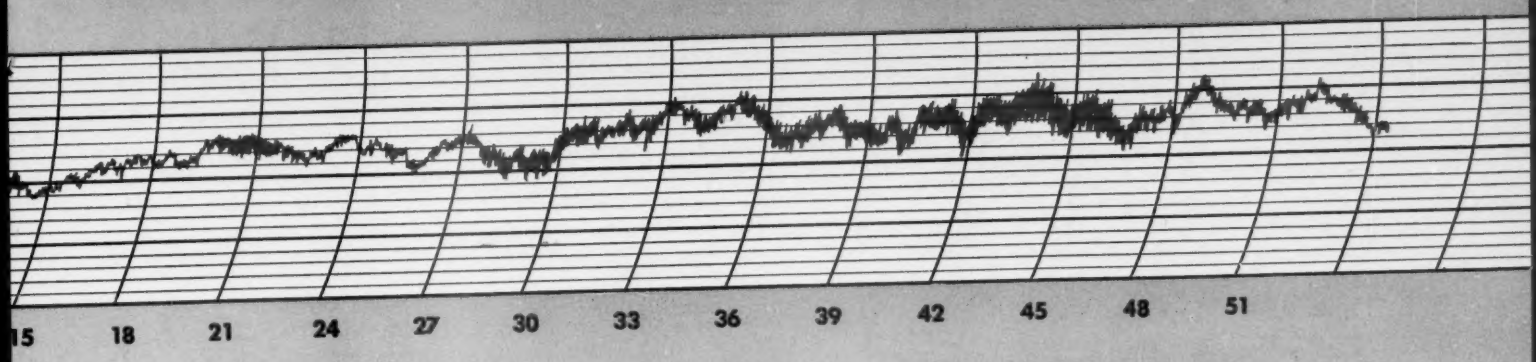
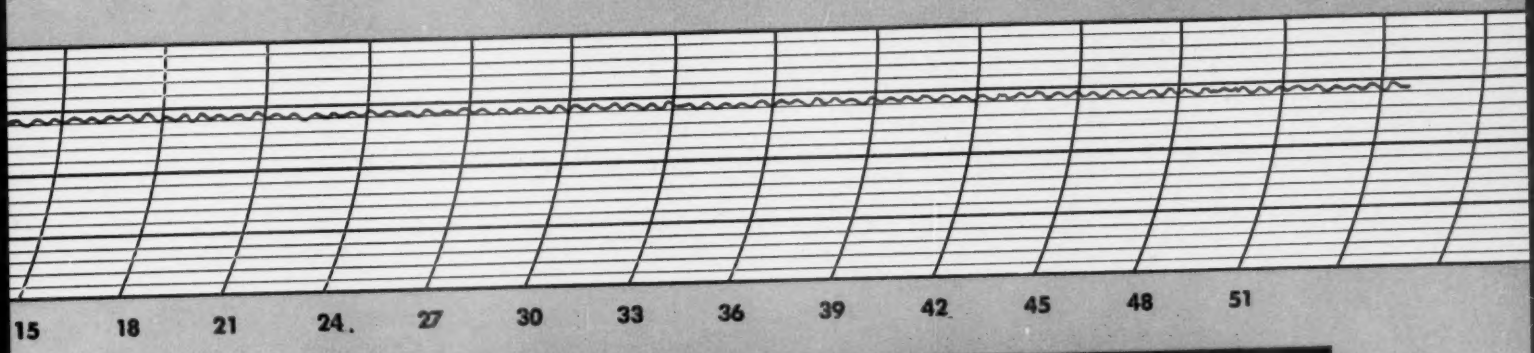


MICROGAUGE SCANS ENTIRE LENGTH OF SUTURE



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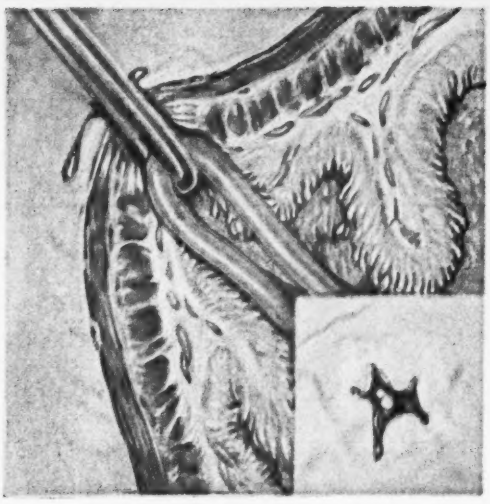
ATRALOC NEEDLES

for minimal suture trauma



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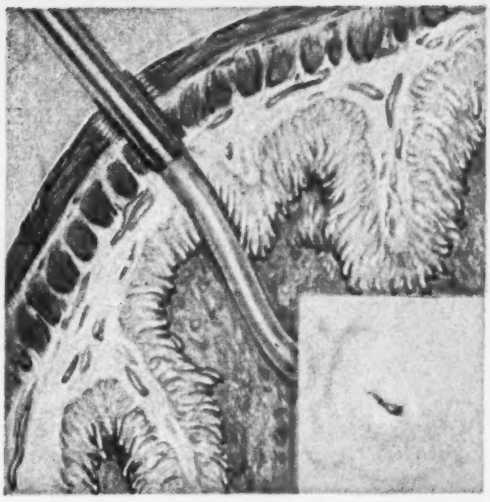

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NEW SERIES VOL. LXIII JANUARY, 1944 NUMBER ONE

Editorial

AN ASCITIC FLUID BANK

FOLLOWING reports of the use of ascitic fluid as a substitute for whole blood we determined early in 1940 to inaugurate an ascitic fluid bank as a majority of our patients as well as donors available were markedly anemic with hemoglobin averages of 45 to 60 per cent (Salhi) and a constant supply of ascitic fluid was always available from cirrhosis cases in the hospital. Davis and White had found ascitic fluid contained a total protein of 2.0 to 3.19 Gm. per 100 cc. of ascitic fluid; albumin values ranged from 0.96 to 1.9 Gm. per 100 cc. with globulin of 0.6 to 0.8 Gm. per 100 cc. of ascitic fluid. The nonprotein nitrogen was reported as 18 to 40 mg. with sodium chloride values of 700 to 750 mg. and calcium of 6.8 to 7.8 mg. We conducted similar studies which disclosed approximately the same values with protein values as high as 4.2 Gm. per 100 cc. of fluid. Davis and White and Choisser and Ramsey reported that ascitic fluid was efficacious in maintaining the systolic blood pressure in dogs in mild and moderately severe shock. No albuminuria, glycosuria or hematuria following its use was noted. Infarction in the brain and lung with death was noted in dogs by Choisser and Ramsey with the use of incompatible fluid and both groups of observers felt cross-matching was necessary because of agglutinins present. We soon observed considerable clotting of fibrin took place in ascitic fluid and believed that infarcts in these cases might be due to fibrin emboli.

The usual Kahn, cultures and, for a time, cross-matching was done on all ascitic fluid used. Repeated filtration or the addition of 50 cc. of 5 per cent sodium citrate per 1,000 cc. of ascitic fluid is necessary to prevent fibrin clots acting as emboli. The transfusing of incompatible ascitic fluid diluted with equal parts of saline without reaction and Wiener's statement that absorption of the donor's agglutinins by tissue cells of the recipient and neutralization by the recipient's own serum will tend to prevent reactions in plasma transfusions led us to use unmatched, undiluted ascitic fluid in a majority of our transfusions. It can be stated it is not necessary to match ascitic fluid as pooled ascitic fluid agglutination titer is low due to neutralization of various iso-agglutinins from different donors. The serum of the recipient has the power of neutralizing agglutinins and the body tissue cells have the property of also neutralizing agglutinins when the titer is low as it is in pooled ascitic fluid. Hemolytic reactions would not be anticipated in ascitic fluid transfusions as Wiener has pointed out the antigens involved in fatal hemolytic reactions are in the red blood cells and not in

* Work done at The Miraj Medical Center, Miraj, India.

the plasma. In over 300 ascitic fluid transfusions in humans we have not noted evidence of hemolytic reactions.

It has been stated that it was not known whether retransfusion after an incubation period of three to five weeks would produce shock in using ascitic fluid. We determined to test this in a series of patients of which thirty were successfully followed. Twenty patients were given intradermal tests with pooled ascitic fluid and transfusions of 300 cc. of pooled ascitic fluid. After a varying period of three to six weeks they were again intradermally tested and again given transfusions of 200 cc. of the same pooled ascitic fluid. The fluid each time was given cold and by the drip method. None of these patients had reactions or showed any evidence of sensitivity such as urticaria, asthma or diarrhea nor was any renal irritation noted. Ten other patients were given transfusions in an identical manner using ascitic fluid removed from the same patient at the time of the original transfusion. No sensitivity was noted; in one patient a chill and slight albuminuria was noted but this was no more severe than was noted at times with glucose. Zimmerman, Strauss and Laufman have recently pointed out that allergic reactions may occur in transfusions of blood of patients with a history of allergy; the same is probably true of ascitic fluid.

In our experience with an ascitic fluid bank we have seen several cases of ascites in which the ascitic fluid was bloody. It should be emphasized that such fluid is not acceptable for an ascitic fluid bank as hemolysis of cells may have already taken place. In addition all of the donors of such fluid we have seen have either had malignancies or marked lowering of the prothrombin time or both; they have failed to respond to adequate amounts of vitamin K.

In over 300 ascitic fluid transfusions from an ascitic fluid bank we have found ascitic fluid is a fairly satisfactory substitute for blood or plasma in the treatment and prevention of shock. Approximately twice as much ascitic fluid is used in a case as would be used if whole blood were being used.

It is not necessary to cross match ascitic fluid as the agglutination titer in pooled ascitic fluid is low and these agglutinins are sufficiently neutralized by the serum and body cells of the recipient to prevent reactions.

Bloody ascitic fluid should not be used in an ascitic fluid bank. The donor should be suspected of either malignancy or prothrombin deficiency or both. In our experience these donors have failed to respond to adequate amounts of vitamin K.

Richard E. Strain, M.D.

Original Articles

INVAGINATION OPERATION FOR ESOPHAGEAL DIVERTICULUM

DONALD E. ROSS, M.D.
Surgical Staff of the Ross-Loos Medical Group
LOS ANGELES, CALIFORNIA

THE invagination method of operation is not new. Girard[1] used it extensively in France. Others in this country have tried it. Why it has not become popular and in general use is difficult to understand. The time honored method of extirpation in one or two stages carries with it the danger of leakage and infection even in the best of hands. If the facts were known, the vast majority of surgeons experience more difficulty from these complications than the published reports would lead us to believe. The invagination method carries with it much less danger as the sac is not opened. If the closing stitches are placed carefully and include only muscle layers, there should never be any leakage. The operation seems much more applicable for use by our vast group of surgeons. It is also very rare that it may not be used. Even large sacs become small ones when the overlying fascia is removed.

History of Early Operations. Nicoladoni, who was the first to operate on a diverticulum in Vienna in 1877, created a fistulous opening. Wheeler, in 1886, performed the first successful removal. Stetten, in 1910, reported forty-eight cases with a mortality rate of 18.7 per cent. It is believed that infection was primarily responsible for this high rate.

Embryology and Pathology. (1) the Pulsion type are more properly called pharyngeal diverticula since they herniate through strands of pharyngeal muscle. (2) Traction diverticula occur in the mid-thorax and usually empty well, as the orifice is at a more dependent position than the sac. (3) Esophageal diverticula also occur at the lower end of the esophagus. This type will not come within the scope of this paper.

Etiology. Moersch and Judd[3] stated that goiter might have an influence. In our three cases one patient had a previous thyroidectomy and a second patient had a large substernal goiter as a complication.

Jackson[2] believed there was an incoordination in the neuromuscular mechanism which results in failure of the lower part of the constrictor muscle to open when a bolus of food arrives at this point. This momentarily causes a great increase in pressure. Some patients give a history of some difficulty in swallowing since early childhood.

Mosher[4] believes there is an embryological background since similar pouches are present normally in some animals, notably the pig. Here there are two pouches, an upper and a lower, but only the lower persists. It may be noted that there may normally be an asymmetry in the pharynx, the pyriform fossa, for example, may be absent. This theoretically might put swallowing reflex off center resulting in strain.

Pathology. In the case of pharyngoesophageal type of diverticulum the sac consists chiefly of mucous membrane covered by areolar tissue which has herniated out between the muscular fibers. Small blood vessels may extend onto the sac. The diverticulum makes its exit posteriorly

but nearly always extends out toward the left side and downward along the esophagus. As a rule the sac does not create a swelling which is visible in the neck but may do so. Also it may be seen on the right side, but even then it is best approached from the left.

FIG. 1. A, this illustrates the position of the incision. B, shows the line of incision of the fascia. C, shows the thyroid with its vascular connections divided and retracted, bringing the diverticulum into view at a deeper level.

In some instances it may be bound to the esophagus or to the thyroid lobe by loose areolar tissue. It is stated that there is difficulty in knowing exactly where the sac begins and where the dissection between esophagus and the sac should be made. This is not true in my experience, the sac being fairly easy to distinguish. A very good way to facilitate this is to have the patient swallow some water at the time of the operation. As a rule this distends the sac readily as a rubber bag would when filled with a heavy fluid. When the sac is emptied by pressure, it contracts to only a fraction of its size, showing a marked degree of elasticity. As a rule there is a slight deviation of the esophagus at the neck of the sac so that when food is swallowed it seems to drop into the sac more readily than it does into the esophagus. There may also be a stricture or considerable narrowing of the esophagus at this particular point. Some authors have, therefore, suggested the patient's swallowing a thread along which a stomach tube is threaded. This more definitely outlines this area at the time of operation.

After the muscle fibers have been peeled off the base and the small blood vessels caught and tied, the sac, being of an elastic nature, has a tendency to contract. This property of the sac explains its contraction after it has been invaginated into the lumen of the pharynx. It shrinks readily and causes no trouble or harm and does not act as a foreign body.

Symptoms and Signs. Typical symptoms are dysphagia, regurgitation of food, an irritable cough, hoarseness sometimes, and in many cases, particularly in old men, there is loss of weight. A large amount of salivation is also present in most cases.

Often food remains in the sac and putrefies, causing a foul odor to the breath; this complaint alone may bring the patient to the doctor. The food seems to drop into the sac first and if this is large, it may twist the upper end of the esophagus so that swallowing is difficult. It may take a long time for the patient to swallow his meal and inanition may result.

Diagnosis. The most important method of diagnosis is by *x*-ray, having the patient stand while some barium solution is being swallowed. Thus the filling of the sac can readily be seen. It is advisable to follow the descent of the barium into the stomach and be sure there are not other diverticula or disorders lower down.

Esophagoscopy is of some value but this, as a rule, yields less information than does the *x*-ray picture. Sometimes a lump is felt in the neck to the left of the esophagus, but this is unusual. Where a stricture is suspected, the extent of this must be investigated and estimated.

Indications for Operation. The chronic irritation and coughing which produces restlessness, loss of sleep and loss of weight are the chief indications for surgery.

OPERATIONS

The Usual One-stage Operation. The danger of operation is chiefly that of infection in the deep fascial planes of the neck. It may be recalled that these fascial planes run down into the mediastinum without any anatomical barrier, and if there is leakage from the operative site there may be widespread infection which may travel downward along these planes and cause generalized infection in the mediastinum with death to the patient.

Fig. 2. This illustrates the further dissection of the diverticulum.

The Two-stage Operation. For the surgeon who operates these cases only occasionally it is much safer to use the two-stage operation. The first procedure in this type of operation is to free the sac and elevate it so that it drains by gravity, packing around the sac with gauze to stimulate the formation of adhesions in the fascial planes of the neck. Occasionally, this is all that is necessary. The sac in these cases collapses and remains empty, causing no symptoms. In most cases, however, it is advisable to operate again, free the sac and cut it off and suture the base or neck of the sac, and in addition repair the muscular coat over the hernial orifice.

Operation by Invagination (Recommended by the author). Anesthesia is accomplished by blocking off the skin with 1 per cent novacaine and deep injection along the posterior border of the sternomastoid muscle.

An incision about four inches in length is made along the medial border of the sternomastoid muscle from the sternal notch upward and outward. The dissection then is carried between the sternomastoid and the infrahyoid group of muscles and these are retracted exposing the lateral border of the thyroid gland. The middle thyroid vein and the inferior thyroid artery are ligated and divided. This permits retraction medially of the thyroid gland and the carotid artery is retracted laterally. This exposes the esophagus. The sac of the diverticulum is then seen running down along the esophagus. The author has not experienced any difficulty in locating this and bringing it up out of its bed with blunt dissection. The sac is seen to be covered with a thin layer of fascia which is stripped off with gauze dissection. A few small blood vessels run up into the neck of the sac. These are ligated and divided. The neck of the sac is cleaned with gauze to outline the muscle edges. These are seen to be quite clear cut. The sac which has now contracted down to a mere fraction of its former size is invaginated into the pharynx and held there while the opening in the muscle is closed with interrupted sutures of silk. These are reinforced

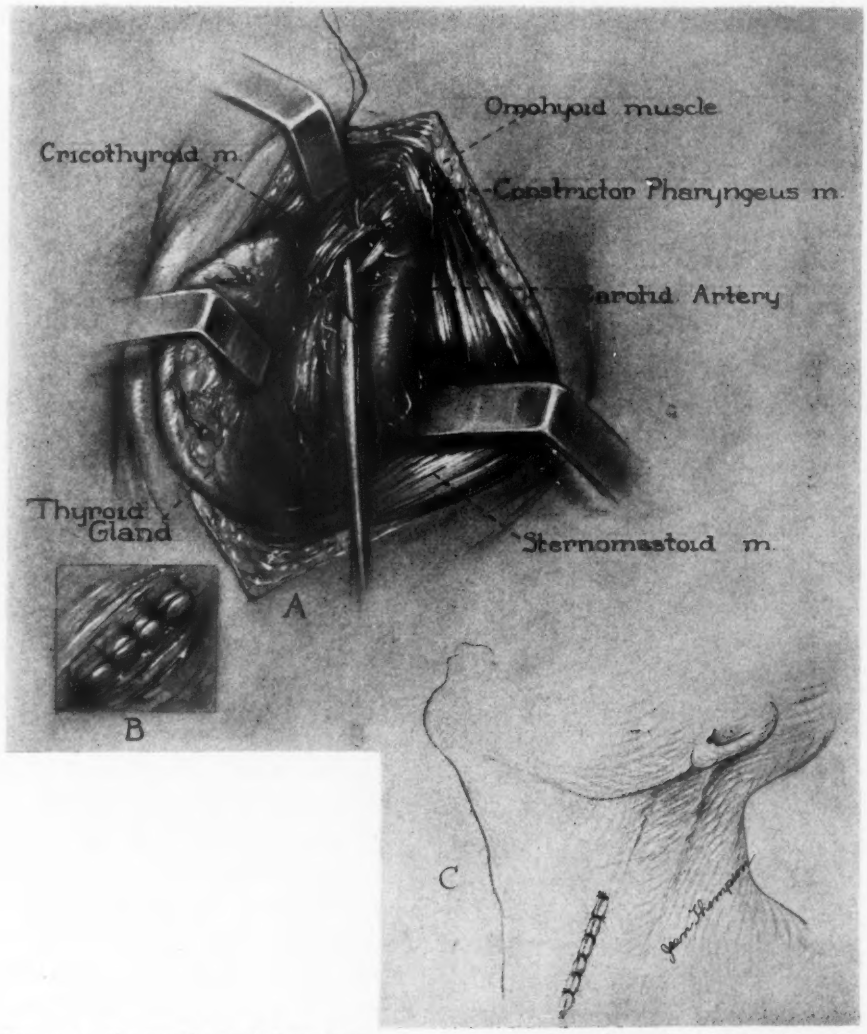

FIG. 3. A, the esophageal diverticulum has now been invaginated and is held in this position by the hemostat. The break in the pharyngeal muscle is shown being repaired by interrupted sutures of silk. B, illustrates the wound closure. C, illustrates the wound closure.

with a layer of fascia sutured over the area. If care is exercised in suturing, the mucous membrane should not be penetrated and there should be little or no cause for post-operative leakage. A small drain is left, however, as a precautionary measure although no drainage has been experienced by us. The fascia of the neck is closed by No. 000 chromic catgut and the skin with clips.

Fig. 4. Case 1. Lateral view before operation.

Fig. 5. Case 1. Lateral view after operation.

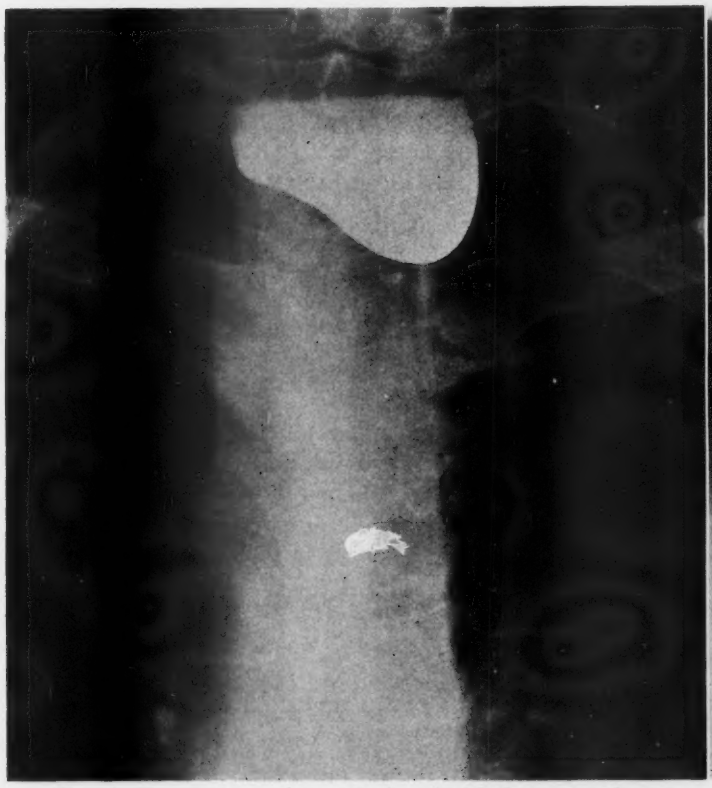

Fig. 6. Case 11. Anterior-posterior view before operation.

Fig. 7. Case 11. Lateral view before operation.

DISCUSSION

The author does not claim any originality in this operation. He does believe that it is applicable to the vast majority of cases. Even large sacs become very small ones when emptied and freed from the surrounding layer of fascia. The contractile power is so great that it is surprising to what extent shrinking will occur. It has previously been felt necessary to do something with the sac. McClure[5] reported a case in which he removed the invaginated sac by snare through the mouth. The author described holding the sac up in the pharynx with a nasal tube for a few days. It is now considered unnecessary to do anything with the sac. It is not interpreted by the patient as a foreign body and causes no trouble. As viewed with a laryngoscope it is seen at first as a redundant fold of mucous membrane and in a few weeks it has a normal appearance.

The immediate postoperative recovery is exceedingly rapid. The operation causes very little general or local reaction as compared with other operations. The patient drinks fluids after forty-eight hours, and the Reyfus tube is removed in thirty-six hours. There has been no recurrence in our cases.

FIG. 8. CASE III. Lateral view before operation.

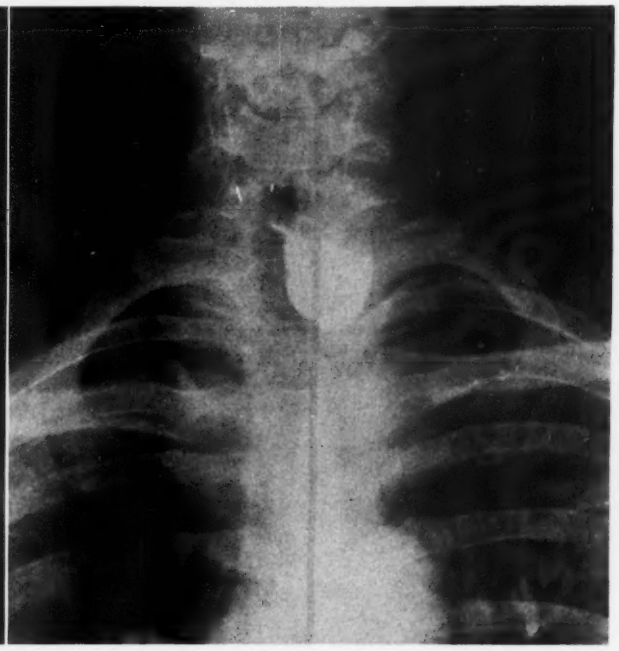

FIG. 9. CASE III. Anterior-posterior view before operation.

CASE REPORTS

CASE I. A male, age fifty-nine, was operated upon in 1932. This patient was operated upon four years before for thyroidectomy.

His chief complaints were irritation in the throat and some difficulty in swallowing with loss of sleep from coughing due to spilling of contents of the sac. Diagnosis of esophageal diverticulum was made by *x*-ray. The operation was performed by invagination, but at that time the sac was held up in the throat for a few days by a catheter which had been tied into the sac at operation. Now, we do not believe that this procedure is necessary.

The operation was performed ten years ago, and there has been complete relief of symptoms and no return of the diverticulum.

CASE II. A male, age fifty-nine, complained of coughing at night, difficulty in swallowing and loss of weight. The diagnosis was corroborated by *x*-ray. The operation was performed April 14, 1942, and was complicated by a large substernal goiter which extended into the thorax at least three inches below the clavicle. This was brought up with some considerable difficulty, and the lobe was removed. Then the

diverticulum was dealt with. The sac was at least two and one-half inches in diameter. When the overlying fascia was removed, the sac was seen to be thin and elastic and contracted down to about the size of a grape. Invagination was done, and the orifice sutured with interrupted silk. A nasal tube was passed down into the stomach and was left in place for forty-eight hours. Convalescence was rapid and the patient was dismissed from the hospital in five days.

He has had complete relief of symptoms. Direct examination through the mouth showed at first only a little fold of edematous mucous membrane and a later examination revealed a normal appearance.

Case III. A male, age forty-seven, was operated upon September 30, 1942. His chief complaint was difficulty in swallowing. The patient had to take a long time with his meal and was annoyed by the spilling over of the contents of the sac. No lump could be palpated in the neck, but the sac showed plainly when barium was taken and the fluoroscope was used.

At operation the sac was nearly two inches in diameter and seemed rather pharyngeal than esophageal. In other words, it arose at a slightly higher level than our other cases and the wall was slightly thicker. However, invagination of the sac and suture of the hiatus in the constrictor was done.

The patient made an uninterrupted recovery and left the hospital in five days. This patient complained somewhat of difficulty in swallowing for a few days, but this rapidly improved and two weeks postoperatively all symptoms had disappeared. Again direct visualization showed that the invaginated sac had contracted so that only a small redundant fold remained. It is believed that it will contract still farther.

SUMMARY

1. The author has attempted to review briefly the salient features of esophageal diverticula.

2. Case reports are hereby given illustrating a method for inversion of the sac and repair of the orifice.

3. This operation is recommended by the author because: (1) It allows the patient to be operated on at one stage; (2) the sac is not opened so that the danger of infection is minimized; (3) the natural contractile character of the sac causes it to shrink and causes no trouble by its presence in the pharynx; (4) it adds another method of operating which may be used to advantage in selected cases, and (5) our patients have had very satisfactory results and continue to enjoy perfect health. The author, therefore, recommends this procedure.

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RECOGNITION AND MANAGEMENT OF BRAIN ABSCESS*

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SEVERAL classifications of brain abscess have, from time to time, appeared in medical literature. A useful one relates to etiology: (1) Secondary to nasal or paranasal sinus disease; (2) secondary to ear and mastoid disease; (3) subsequent to compound wounds of the head, particularly when the dura has been lacerated; (4) "metastatic" brain abscess from suppurative processes elsewhere in the body, such as: chronic osteomyelitis of the long bones, lung abscess, bronchiectasis, etc. Undoubtedly, the first two etiological factors are by far the most important from the standpoint of frequency and as sources of the more surgically favorable abscesses.

A brain abscess may be located in the cerebrum, the cerebellum or the brain stem. This paper concerns itself only with those in the cerebrum and cerebellum proper and does not include the extradural and subdural abscesses which are not *true* abscesses in the surgical definition of the term, i.e., an infection which is circumscribed or walled-off and surrounded by a more or less well defined capsule.

HISTORY

The history in a case of brain abscess is often of the greatest importance, but, occasionally, a record of previous infection is not given the weight it should have in the differential diagnosis because at the time the patient first comes under observation a previous infection may be quiescent or seemingly "cured." One such case, seen several years ago at the University of Virginia Hospital in Charlottesville, was that of a white boy of ten years who had had a right-sided otitis media without a mastoid operation three months before admission to the hospital. Paracentesis of the right ear drum had been done and one of the sulfonamides had been administered. The ear infection subsided; the temperature became normal, and the boy appeared well until about two weeks before his hospital admission when drowsiness supervened together with headache and vomiting. There was no drainage from either ear at that time. The child had a marked ataxia of the extremities on the right side and a high degree of choked disk bilaterally. The temperature was subnormal, and the pulse rate was 50 to 60 per minute. A cerebellar tumor was presumed to be more likely than abscess, and a bilateral cerebellar exploration was carried out after a ventriculogram had disclosed an internal hydrocephalus of marked degree. A large abscess was encountered and drained in the right cerebellar hemisphere near the cortex, marsupialization being carried out. The child eventually recovered after a prolonged convalescence due to a persistent cerebrospinal fluid fistula which finally could be closed.

A history of head trauma and of ear and sinus infections (frequently mild) is often obtained in cases of brain tumor (demonstrated at operation) and proves to be of no clinical importance. Relatives and friends of the patient often prefer to believe that such an inciting factor (particularly trauma) is the real cause of the symptoms. On the other hand, a history of a fairly recent head injury, especially one involving the paranasal sinuses or the mastoid, should be given careful consideration when the diagnosis is considered in a patient with

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increased intracranial pressure with reference to the possible presence of a brain abscess. Months may elapse after such an injury before signs of increased intracranial pressure develop due to a brain abscess. It is most important, *surgically*, to suspect an abscess preoperatively, when it is present, as such a lesion, in contrast to intracranial tumors, is usually dealt with through a small burr opening rather than an osteoplastic flap or suboccipital craniectomy, to prevent spread of infectious material over the cortex of the brain and into the subarachnoid space.

All compound depressed skull fractures in which the dura and the brain cortex have been lacerated are potential cases of brain abscess postoperatively, even in this day of early operations after head injury and the use of sulfonamides. If signs of increased intracranial pressure develop weeks or months after an operative repair of a compound skull fracture with dural laceration, an abscess must be considered. Such a complication is, however, a remarkably infrequent occurrence in such cases, provided the primary operation has been properly performed a few hours after trauma.

In regard to a "metastatic" abscess from distant foci in the body, every patient who has had a boil or carbuncle, chronic osteomyelitis of the long bones, a lung abscess or bronchiectasis is a possible candidate for a brain abscess, just as a *spinal* extradural abscess may develop from the same sources.

PATHOLOGICAL ANATOMY—PATHOGENESIS

Concerning the paths of infection into the meninges from the nose and ear, Eagleton,[1] in an extensive review of the subject, quotes Rosenow's[2] careful experimental work which demonstrates that trauma, operative or otherwise, produced in a patient who is the host of a quiescent infectious lesion, such as a nasal sinus or mild chronic ear infection, can institute a bloodstream invasion, which, if the microorganism has a selective action on the central nervous system, may cause a grave infection. From the nose, infection can travel to the frontal lobe by way of the arachnoid prolongations surrounding the olfactory nerve. Developmentally, there is no evidence, according to Weed and Sabine,[3] that the lymphatics extend into the central nervous system, but the arachnoid prolongations along the olfactory nerve have a communication in the mucous membranes of the nose with the lymphatic system. Sphenoid disease can involve the supra-adjacent basilar cisternae through the periosteum and bony wall of the sphenoid sinus.

Infection in the nose and ear can also be transmitted to the brain by venous extension. It has been demonstrated that the venous circulation of the tympanic cavity is in direct communication with the inferior petrosal sinus, independent of the internal auditory veins; and there also exist direct venous relations between the tympanic cavity and the apex of the petrous portion of the temporal bone. In sphenoid and other sinus diseases, osteomyelitis of the sinus wall and adjacent skull can occur from a primary infection of the mucous membrane of the sinus, thence along the Haversian spaces to the diploë of the osseous wall of the sinus and, occasionally, to the cavernous and other venous sinuses.

Bloodstream infection and transmission is undoubtedly the mechanism for the production of brain abscess from a distant focus as in osteomyelitis of the long bones, etc., and direct inoculation infection is the mode of entry in brain abscesses following compound head wounds, especially in those in which the dura and the cerebral cortex have been lacerated at the time of trauma.

It is the ability or failure of the host to form a capsule about the more or less localized brain infection that makes for success or failure, in large measure, in the operative attack upon any individual case of brain abscess. Otogenic brain abscesses resulting from otitis media and mastoiditis

are in the majority, and extension from these sources, most frequently, involves the temporosphenoidal lobe (cerebrum) and the cerebellum. In one series of cases quoted by King,[4] 83.2 per cent of a series of brain abscesses arose primarily from an otitis media and mastoiditis, and 9.1 per cent from infections of the nasal cavity and its accessory sinuses. The spread of infection to the brain from the middle ear and from mastoiditis, by direct extension, is through purulent thrombosis of the lateral sinus, osteomyelitis of the tympanic wall, along the adventitial spaces of perforating blood vessels, and through the internal auditory meatus. The spread of infection from the nasal cavity and its accessory sinuses, by direct extension, is due to osteomyelitis of the sinus wall, along the perineural spaces, and along and through the veins.

Another rarer source of brain and subdural abscess is in the patient with a skin malignancy of the scalp, of, perhaps, many years' duration, that fails to heal entirely in spite of surgical excision and *x*-ray or radium therapy. Signs of infection (chills, high fever, etc.) and/or increased intracranial pressure and, occasionally, convulsions herald the invasion of the meninges and/or the brain itself from the original external infection.

Regardless of the mode of infection, a brain abscess commences with an inflammatory leucocytic infiltration and actual softening of the subcortical brain substance and terminates by the conversion of a portion of the brain into a cavity containing pus. In the acute form the abscess has no definite wall. It consists of a broken down area of brain tissue, which might be termed acute suppurative encephalitis, with a central area containing pus and necrotic material from degeneration of the subcortical white matter. The wall, at first, is irregular and ill-defined and consists of brain tissue with a shaggy surface, surrounded by an area of edematous brain substance. Should the patient be fortunate, this process becomes chronic in that a well defined wall or capsule is established, and the abscess then truly follows the old surgical description of an abscess as being a "circumscribed cavity containing pus." The wall of the abscess consists of an internal layer composed of purulent tissue, the so-called "pyogenic membrane," and an external layer of granulation tissue composed of various proportions of fibrous macroglia and fibrous tissue derived from the vascular adventitia and, in some instances, from the leptomeninges. The granulation tissue is infiltrated with plasma cells, lymphocytes, and a few eosinophiles. The infiltration is continued in the adventitia of the vessels for a considerable distance into the brain.

It has not been determined accurately how long it takes for a definite wall or capsule to form around a brain abscess. Penfield[5] stated that the wall of an abscess begins to form in the first week after invasion of the brain, but that it does not offer resistance to a brain cannula until two to three weeks have elapsed.

Grant[6] has noted that a definite capsule or firm wall is well established in the interval between the fourth and sixth weeks after invasion of the brain, and Bagley[7] has shown a section of an abscess wall which formed in three or four weeks. Bagley has stated that the thickness and firmness of the abscess wall will be determined by the relative amount of mesoblastic and glioblastic fibrous tissue present, the amount of glial tissue in the wall, and the length of time the abscess has existed. If not evacuated in time, the abscess tends to rupture into the subarachnoid space or into an adjoining ventricle with disastrous results (meningitis or purulent ependymitis.

SIGNS AND SYMPTOMS—DIAGNOSIS

The state of consciousness is, perhaps, the most important single observation which the physician can make in a suspected case of brain abscess. If the patient is conscious, well oriented, and alert, no matter how high the fever (frequently in

favorable cases there is little or no pyrexia) or how slow the pulse, an abscess requiring immediate drainage is unlikely, although possible. A choked disk in one or both eyes, sometimes of a high degree, is almost the rule in *favorable* cases of brain abscess; a homolateral third nerve paralysis is frequent in frontal lobe abscess. On the other hand, abscess of the brain is notoriously treacherous and difficult to diagnose and, more than once, to the chagrin of the clinician, the pathologist has revealed, at the postmortem table, a large "silent" brain abscess (perhaps with rupture into the ventricular system) as the cause of the patient's death. The reason for this is that once infection has entered the cerebral substance, it finds itself in tissue lacking lymphatics, so that if the meninges have protected themselves against it, if it is associated with an infected sinus draining externally, or if it does not break into a lateral ventricle, it fails to cause the ordinary signs of infection. Should an abscess form in the temporal or frontal lobes, the most common sites, or in the cerebellum and not, at first, be extensive enough to cause either signs of increased intracranial pressure or localizing symptoms (hemiparesis, aphasia, hemianopsia, Jacksonian convulsions, unilateral ataxia, etc.) it may long escape notice. Accordingly, it enlarges in spite of the fairly vigorous reaction and encapsulation with which the brain responds to it, until finally it causes pressure symptoms (headache, bradycardia, stupor, choked disk, and possibly even the usual signs of deep penetration into the temporal lobe, i.e., an homonymous hemianopsia). If situated on the left side in a right-handed patient, it may cause aphasia. With deep involvement of the cerebellum, nystagmus and ipsolateral ataxia will usually be provoked together with high-grade choked disks.

In a well encapsulated abscess of long standing, marked bradycardia of fifty to sixty beats per minute and hypothermia (95° to 96°) are characteristic, even more so than with intracranial tumors. In cerebellar abscess, the ipsolateral ataxia of the extremities is striking and more clear-cut than in most cases with cerebellar tumor.

Given a patient with a history of a definite infection adjacent to the head or remote from it in the recent past, who develops signs of increased intracranial pressure associated with stupor and choked disks with or without localizing neurological signs, a brain abscess must be diagnosed clinically and operation promptly carried out. A lumbar puncture in such cases is usually to be avoided, particularly if choked disk is present. A ventriculogram for precise localization may be necessary in the absence of localizing signs. Cases of primary frank pyogenic or meningococcic meningitis rarely, for some reason, develop brain abscesses of any appreciable size or of surgical significance, possibly (in the fatal cases) because death occurs too soon after the meningitis develops.

As in the diagnosis of many lesions in any branch of medicine, it is the "awareness" of the diagnostician to the possibility of an abscess being present that makes for success in the recognition of these treacherous lesions so that operation will be carried out before profound coma develops in which condition operation is, as a rule, of little or no avail.

TREATMENT

Treatment consists of evacuation and adequate drainage of the abscess at the optimum time. There are several acceptable methods utilized in various clinics: (1) Drainage of the abscess through a small trephine opening in the skull and immediate or delayed insertion of a soft rubber catheter into the abscess, which is left *in situ* usually from four to six weeks. (2) In certain rare instances, according to Cairns and Donald,[8] thickwalled, multilocular abscesses cannot be treated satisfactorily by drainage but require dissection and total removal or marsupialization. Abscesses in which the pus is thick are probably best treated through an osteoplastic flap, ac-

cording to these authors. (3) Single or repeated tapping of the abscess without drainage of any kind as recommended by Dandy[9] who believes that in an encapsulated abscess the brain needs only a relief of tension to enable it to overcome the infection. (4) Unroofing or marsupialization of the abscess, i.e., removing the overlying brain cortex which covers the abscess, as proposed by King[10] in 1924, and in 1934 and 1938 by Horrax[11] (after Cushing). King stated that by his method (marsupialization) the abscess is everted and obliterated by the associated increased intracranial pressure, which is in fact the case; but this last method is of value only in quite superficial well encapsulated abscesses of many weeks' standing, preferably located in the anterior portions of the frontal lobes, remote from the motor cortex.

The essential problem in the surgical management of brain abscess is to wait long enough for the abscess to encapsulate, but not to postpone operation to the point that rupture into a ventricle or the subarachnoid space is imminent or has already occurred. The finest surgical judgment is required in determining when to operate in any individual case.

MORTALITY AND RESULTS

It must be recognized that certain abscesses are inherently unfavorable from the beginning of their existence. In such a group of cases the mortality is practically 100 per cent. This fundamental point should be kept in mind in considering the mortality percentages quoted from any clinic, as some reports include all types of brain abscess; others discuss only the *favorable* type. In multiple brain abscesses and in poorly defined abscesses with little or no capsule formation, but with marked edema and purulent encephalitis about the abscess, a fatality is almost to be expected and can scarcely be avoided by any operative procedure. In some of the deep abscesses (5 to 6 cm. or more beneath the cortex) of the cerebral hemispheres, a fatality occurs due to difficulties in accomplishing adequate drainage and inoculation of adjacent white matter in attempts to establish drainage. *It is with the more or less superficial cerebral and cerebellar abscesses with at least fairly well defined capsules that operation at the optimum time is life-saving.* In 1927, Dr. Coleman[12] reported a series of fourteen patients with chronic brain abscesses, twelve of whom recovered. Simple tapping and drainage with a soft rubber catheter was used in each case. All of these abscesses were in the cerebral hemispheres, being located in the temporal, frontal, and parietal lobes. At a later date, he[13] recorded a series of twenty-eight cases of brain abscess with twenty-one recoveries, drainage being accomplished very simply and adequately in each case with a small soft rubber catheter or silver cannula. Horrax,[11] in 1938, published a study of thirty patients with brain abscesses (favorable and unfavorable) treated by him during a period of nine years. He stated that, in the *favorable* group, the prognosis should be good (fifteen of eighteen patients recovered) with a mortality rate not exceeding 10 to 20 per cent, instead of the generally accepted death rate of 30 to 40 per cent. This desired mortality percentage (10 to 20 per cent) in the so-called favorable group is probably concurred in by most neurosurgeons today.

Quite recently (1942) Grant[14] has collected a series of one hundred cases of brain abscess observed at the University of Pennsylvania Hospital. Fifty-three patients (53 per cent) died, but it should be stated that a fair percentage of those patients were of the unfavorable group, i.e., (1) multiple abscesses of the brain, (2) poorly defined or non-encapsulated abscesses, (3) cases having proved bacterial meningitis at the time of operation as demonstrated by lumbar puncture before drainage of the abscess. Grant found the postoperative mortality for closed drainage and open drainage to be the same (33 per cent) in his series of cases.

All neurosurgeons agree that, if possible, a brain abscess should not be attacked until encapsulation has occurred. Three to four weeks from the onset of intracranial symptoms, therefore, should be permitted to elapse before the purulent cavity is evacuated, although, that certain abscesses will *never* become well encapsulated is frankly conceded; and indeed sometimes the surgeon is forced to operate before that time, chiefly because of developing stupor, convulsions, or marked bradycardia associated perhaps with severe headache and vomiting.

We believe that the initial attack upon a brain abscess usually should be carried out through a small trephine opening directly over the lesion (a previous ventriculogram may be necessary) with the insertion of a drainage tube (French rubber catheter) whether the abscess is in the cerebellum or the cerebrum. This procedure alone will result in a cure in many instances, the tube being left *in situ* for several weeks. If this method does not produce satisfactory results, a resort must be had to more radical methods (? of marsupialization) involving the possibility of more serious neurologic sequellae (convulsions, hemiparesis, aphasia, etc.) after recovery from the infection has occurred.

CONCLUSIONS (AS RELATED TO THERAPY)

Conservative surgical treatment, i.e., tapping alone or tapping with catheter drainage through a small trephine opening in the skull produces the best results in most cases in the treatment of brain abscess. The use of wide-open drainage has not lowered the operative mortality in comparison with more conservative methods (Grant). In follow-up examinations, too, a much higher percentage of patients are returned to their former occupations following the use of the simpler procedures for instituting drainage than when a more radical technic involving wide destruction or removal of overlying brain tissue is employed. Furthermore, the less radical the surgical treatment of a brain abscess, the less likely are *convulsions* to occur months or even years after operation, a postoperative complication reported to be as high as 53 per cent in certain clinics.

Needless to add, the appropriate sulfonamide should be administered by mouth or parenterally in rather large doses immediately upon establishing the bacterial diagnosis in any individual case of brain abscess or even before such a diagnosis is made when a previous adjacent or remote infection (the usual course of events) has been treated before intracranial symptoms supervene. Its use locally in or about the brain abscess itself seems to predispose the patient to postoperative convulsions which otherwise might not occur and, for that reason, such drugs are usually not so employed.

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COMPLICATED TRAUMATIC DISLOCATIONS OF THE HIP

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KEY and Conwell state that "Traumatic dislocations of the hip are quite rare and comprise only from two to five per cent of all dislocations, the exact percentage varying slightly in different series. This is in marked contrast to the shoulder where over fifty per cent of all dislocations occur." This largely follows our experience.

The simple, uncomplicated dislocation can usually be reduced by one of the methods in general use—Allis, Leadbetter, Bigelow or Stimson. The cases complicated by fractures, either of the femur or of the acetabulum and pelvis often are difficult to manage. The four cases to be reported here were of the complicated variety and presented problems of treatment which we believed would be of interest.

CASE REPORTS

CASE I. B. W., a white male, forty-four year old painter, fell a distance of thirty feet on April 6, 1942, and was immediately admitted to the Hospital in profound shock. A Thomas splint had been applied to the right leg. Examination showed a deformity at the right hip with the extremity internally rotated and shortened. The splint was retained and the patient treated for shock. Further examination indicated injury to the left side of the chest. *X*-rays revealed depressed fractures of all of the true ribs except the first. The right femur was dislocated posteriorly, being markedly displaced upward and carrying with it a fragment of the roof of the acetabulum.

On account of the serious condition of the patient, no immediate attempt at reduction was made. Russell traction was applied as a temporary measure, and was continued for several days until the patient had recovered from shock. Another *x*-ray was made preliminary to trying reduction. However, this was not necessary as a satisfactory reposition of the dislocation had resulted with the acetabular fragment in a good position. This traction was continued for six weeks, with interval *x*-rays to check position. No serious complications arose from the fractured ribs, except for a moderate degree of subcutaneous emphysema. He was discharged from the hospital after eight weeks, having been out of bed previously for several days.

A caliper brace was procured in the hope of early walking. However, this was delayed on account of deep pressure ulcers on the anterior aspect of the ankle. *X*-rays at discharge showed retention of reduction with the fragment of the acetabulum forming a well designed shelf. Motion in the hip was free and painless. He was able to return to a lighter type of work in October 1942. This patient has now returned to his former type of work. *X*-ray examination in April, 1943, showed no abnormal changes in the head and neck of the femur.

CASE II. M. K., female aged twenty-nine years, a supervising nurse, was injured on April 21, 1942, while driving a car in a nearby town. She was admitted to the hospital twenty-four hours after the accident, with a Thomas splint applied to the left leg and dressings to right foot and leg. Examination showed deformity of the left hip with some internal rotation of the thigh and shortening. There was a marked contusion about the left knee. In the right leg below the knee there was seen a deep wound, and in foot a deformity of the third, fourth and fifth toes. *X*-rays had been made in hospital near the scene of the accident and showed a posterior dislocation of the left hip, accompanied by extensive fractures of the acetabulum and ilium. Toes of the left foot were fractured; examination of the leg was negative.

The patient was prepared for anesthesia and early next day an unsuccessful attempt was made to reduce the dislocation. The Leadbetter method was used but failed because the patient was very muscular and satisfactory relaxation could not be obtained even under deep anes-

Fig. 1. Case 1. B. W. Posterior dislocation with fracture of roof of acetabulum, April 6, 1942.

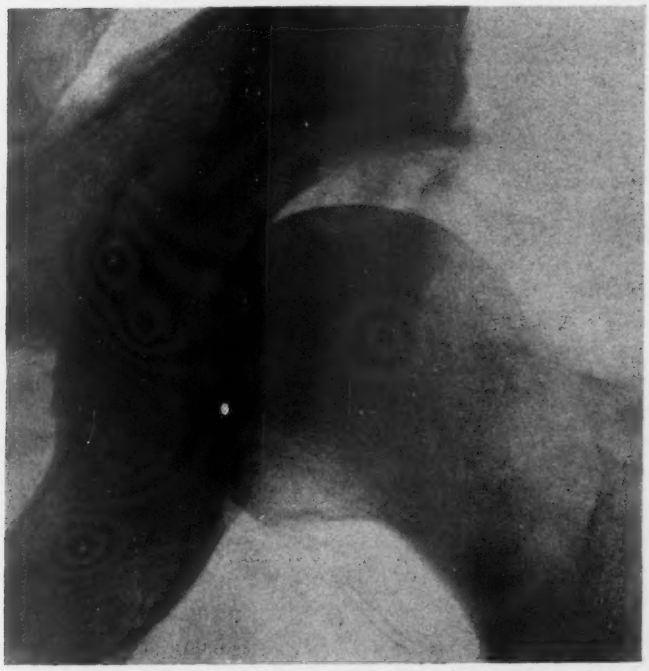

Fig. 2. Case 1. Shows dislocation reduced with fragment forming a shelf above and behind the head of femur, June 30, 1942.

Fig. 3. Case 11. M. K. Posterior dislocation with displaced fracture of acetabulum and ilium, April 22, 1942.

thesia. The fractured toes were reduced and a cast applied. The patient was then returned to bed and Russell traction was applied to the left leg and retained for several days, when another unsuccessful attempt to reduce was made. Caution was used in the force applied because of fear of injury to soft parts, especially the sciatic nerve, since the head of the femur was so grossly displaced posteriorly and pushed upward. Skeletal traction was then instituted by insertion of a steel pin through the lower third of the femur with the hope of overcoming muscle spasm with possible reduction of the dislocation. *X*-ray studies were made every third day to check progress. These showed a gradual pulling down of the femur but with no reduction. Since this was a seriously complicated dislocation with an unfortunate prognosis if left unreduced, open operation was under consideration. As this is a formidable procedure, consultation was requested before coming to a decision. Dr. H. H. Ritter was asked to see the patient and it was then decided to make one more attempt at reposition.

Fig. 4. Case II. Dislocation reduced with improved position of fragments, May 18, 1942.

Under general anesthesia, a strong traction was made and the leg gradually carried into wide abduction at the same time making pressure over the greater trochanter. While this maneuver was being carried out, there was a distinct click as the femoral head was apparently forced into what remained of the acetabular cavity. The leg was then strongly rotated internally and *x*-rays immediately taken showing satisfactory reduction. Traction was maintained in this position with interval *x*-rays for eight weeks. During this period, the patient was encouraged to move the hip carefully, limited by any pain produced. In a few days pain became very slight so that motion could be more active. The final *x*-ray showed maintained reduction with apparent reformation of an acetabulum. A caliper brace was then applied and patient allowed to walk, first with the added support of crutches and later the use of a cane. On August 1, 1942, she returned to her full duties, with motion in hip free and painless. On November 1, 1942, retakes showed the reduction complete and no joint disorder. There was noted a small fragment still free above the acetabulum. At the

FIG. 5. CASE II. Complete reduction maintained with acetabulum well formed and no evidence of pathological disturbance. November 30, 1942.

time of this report, she has dispensed with the use of a cane and carries out all her duties wearing the brace. *X*-ray examination in October, 1942, indicated no abnormal changes. A recent report states that she has had no resulting disability.

Fig. 6. Case II. Lateral view showing acetabulum in better detail, November 30, 1942.

Case III. H. F., a white male, twenty-nine years old, was admitted on May 27, 1942, immediately after a road accident. He was in severe shock. A Thomas splint had been applied to his right leg. There were numerous lacerations about the face and hands and a deep wound in the posterior thoracic region. Marked deformity was noted at the right hip with the thigh internally rotated and apparently shortened. *X*-rays showed a posterior dislocation of the hip with marked displacement combined with extensive fracture of the acetabulum and ilium.

On account of the extreme shock, no immediate attempt at reduction was made. The lacerations were treated and Russell traction applied to the right leg. However, this had to be abandoned within several days on account of skin irritation. The patient's condition had then improved sufficiently to try reduction. There had resulted some improvement from traction and under light general anesthesia on May 31, 1942, reduction followed strong traction, abduction and internal rotation. To maintain this position, skeletal traction was instituted by inserting a steel pin through the lower third of the femur. Interval *x*-rays were made while traction was maintained until July 24, 1942. During this period, active motion of the hip was encouraged. On August 9, 1942, the patient was allowed out of bed on a chair and left the hospital on August 19, 1942, wearing a caliper brace and walking with crutches. His progress was steady so that by October 6, 1942, he was able to return to his work as an electrician. He had no pain or disability in the hip. On November 18, 1942, *x*-ray retake showed reduction maintained with good joint outline and fracture of ilium well healed. At the time of this report, January, 1943, he was at work daily, wearing his brace and had no pain or disability at the hip. This patient has

continued at his former occupation with no disability. Several retake *x*-rays have shown no abnormal changes in the head and neck of the femur.

until the patient had recovered from his shock. On the second day, an attempt at reducing the dislocation was made. Because very definite resistance was encountered while manipulating,

FIG. 7. CASE III. H. F. Posterior dislocation with marked comminuted fracture of acetabulum and displaced fracture of wing of ilium, May 27, 1942.

CASE IV. M. F., a white truck-driver, aged eighteen years, was injured in a road accident on October 28, 1942, sustaining a comminuted fracture of the shaft of the right femur with marked shortening and displacement. There was also marked deformity of the left hip region with the thigh internally rotated and apparently shortened. *X*-rays showed a posterior dislocation of the left hip with a fracture fragment visible in the region of the acetabulum. The roentgenologist was not able to determine definitely the origin of this fragment even after several retakes. The severe shock present prevented any immediate reduction of the fracture or dislocation.

Russell traction was applied to both limbs excessive force was not used for fear of injuring soft parts. No reposition was obtained. Skeletal traction through the lower third of the femur was then instituted. Two additional attempts at reduction at intervals of several days were unsuccessful. It was then decided to continue the skeletal traction for a further period hoping to avoid open operation. Interval *x*-rays indicated that some progress was being made in pulling the femur down, but a final effort to reduce failed. After allowing a period of five days for the parts to recover from the effects of manipulation and to prepare the patient both locally and in general condition, open operation was done. This was three weeks after the accident. Exposure was made by use

of Smith-Petersen incision and showed a marked posterior displacement of the femur with a shell fragment of the head still partially attached and impinged on the upper rim of the acetabulum. This was evidently the condition which prevented reduction by manipulation. After some difficulty the fragment was removed and the acetabulum exposed, removing the remains of the torn ligamentum teres. The hip was then reduced and a cast applied to the entire extremity incorporating the pin which had been inserted for skeletal traction. The patient reacted well and one week later, open reduction was done on the fracture of the right femur, with a cast applied and incorporated in the cast on the left hip. The cast was removed from the left hip eight weeks after operation and active motion encouraged. On account of the fracture of the right femur, it was still necessary to keep him in bed. However, as *x*-rays now (January, 1943) show good position maintained in both areas of injury, it is planned to have him out of bed soon and to supply a caliper brace for his left leg.

Since the above report was made a one inch shortening of the right femur was noted but with no disability in the left hip. *X*-ray examination in June, 1943, showed no abnormal signs in the head and neck of the femur. The patient has been working at a lighter type of work since October 1, 1943. Further *x*-ray studies have been advised.

FIG. 8. CASE III. Dislocation reduced with fractures in improved position and acetabulum reformed, July 15, 1942.

COMMENTS AND CONCLUSIONS

Complicated traumatic dislocations of the hip present serious difficulties in treatment. Attempts to reduce should be made as early as possible, depending on complicating conditions. Force used should be carefully controlled to avoid injury to soft parts, especially the sciatic nerve. If manipulation is unsuccessful, traction either by the Russell method, which may

FIG. 9. CASE III. Reduction maintained with acetabulum well formed and protected by an extensive shelf, November 20, 1942.

Fig. 10. Case iv. M. F. Posterior dislocation with loose fragment apparently in acetabulum, November 15, 1942.

Fig. 11. Case iv. Reduction after operation, December 15, 1942.

be sufficient in some cases, or by skeletal traction through the femur, should be instituted.

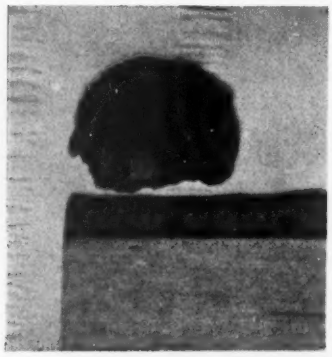

FIG. 12. CASE IV. Fragment removed from posterior rim of acetabulum.

FIG. 13. CASE IV. Severe type of fracture of right femur complicating the dislocation of left hip.

We believe that skeletal traction is preferable, especially in complicated displacements, since by this method one avoids the possibility of pressure ulcerations following prolonged traction, which occurred in our treatment of the patient in Case I. During this period of traction, frequent *x*-rays should be made to check progress and further attempts to reduce should be tried when conditions indicate. If these fail, open operation should follow, preferably within three weeks, as operation becomes increasingly difficult. Following reduction, fixation by cast is indicated in the simpler cases. In others, as in Case II, where there is extensive fracture of the pelvis and acetabulum, skeletal traction should be continued for eight weeks. Active motion of hip and knee should be encour-

aged to the degree possible and should be limited by pain. Numerous *x*-rays are required to follow progress.

even in the simpler cases. In the cases reported here, it is of course too early to determine the end-results, but they will

FIG. 14. CASE IV. Reduction and wiring of fragments.

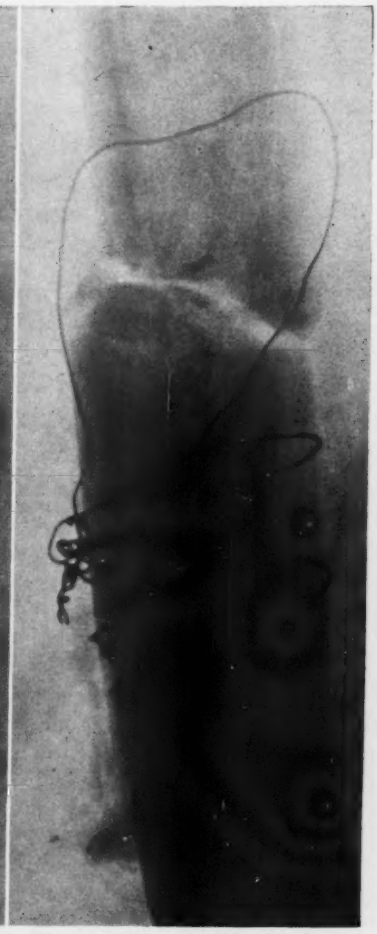

FIG. 15. CASE IV. Lateral view.

We believe that all patients should be fitted with a caliper brace to allow walking without direct stress on the joint, as aseptic necrosis of the head with associated joint changes may follow in a certain number of cases, even if not of a complicated type. These changes are due to the interference with circulation caused by the damage to the ligamentum teres and other structures carrying nutrient circulation. Such disturbances have been known to occur even three to four years after injury. Follow-up care is, therefore, very important, requiring *x*-ray study at certain intervals for early detection of such changes. With these possibilities, prognosis must be guarded be followed, if possible, for at least two years before final evaluation will be attempted.

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THE MANAGEMENT OF VARICOSE VEINS IN ARMY PERSONNEL*

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TREATMENT of varicose veins in the Army presents problems not encountered in civil practice. The first problem is the more difficult differential diagnosis. Pain, aching, and tiredness in the legs following marching is most common among soldiers whether varicose veins are present or not. It is natural for soldiers, especially during the preliminary phases of training, to magnify these symptoms in order to avoid the more arduous work. In addition, a certain by no means small group of psychoneurotic patients combine these symptoms and small varicosities that are present with their many psychoneurotic complaints to produce a confusing clinical picture which will not respond to varicose vein therapy. Second, there is need for a treatment which will be rapid as well as effective. Cure must be permanent if possible. Palliative measures which involve repeated hospitalization or numerous outpatient visits interfere with military training and must be avoided. Patients must be returned to duty able to do full duty.

An effective plan of diagnosis and treatment must provide (1) criteria for selection of cases which require treatment, (2) simple diagnostic maneuvers which can be carried out with the minimum of equipment, and (3) an adequate surgical technic which will allow return to full duty within a short period.

CRITERIA FOR SELECTION OF CASES

Military practice demands that patients operated upon for varicose veins must subsequently be able to do full duty. Operations on soldiers, who are to be reclassified for other conditions, are useless. There are four principal groups of patients that must be excluded in the selection of cases. These are: (1) the psychoneurotic, (2) those with associated orthopedic conditions, (3) those with associated peripheral vascular disease, and (4) those, who for want of a better term, are classified as myalgic asthenia.

Patients with Psychoneurosis. The psychoneurotic patient is a daily problem in a varicose vein clinic. His veins may be mild or severe but his symptoms are manifold. Complaints referable to the legs are many and bitter. But symptoms are by no means confined to the legs. They can and do complain of everything, but most often present headache and dizziness, transient pains of the back, shoulders and neck, palpitation and breathlessness. These patients must definitely not be treated for their varicosities. They should be reclassified to limited duty or discharged from the service for their psychoneurosis. In a busy clinic it is not always easy to segregate these patients. In addition to careful questioning about complaints other than the legs, it is useful to ask two questions: first, how much schooling has the patient had, and second, has the patient ever had a nervous breakdown. Less than fifth grade schooling or positive answers to the second question should prompt a neuropsychiatric investigation before surgery, regardless of the severity of the varicose veins.

Patients with Associated Orthopedic Conditions. Varicose veins are often seen in

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patients with associated orthopedic conditions. A careful evaluation must be made as to whether the symptoms are due to the varicosities or whether they are actually due to the orthopedic disorder. A list of orthopedic diseases seen in patients whose entrance complaints have been referable to varicose veins is imposing. Pes planus, pes cavus, hallux valgus and metatarsalgia have been the most common. Muscle sprains of the calf and pain on marching in the region of the tibialis anticus muscle are seen frequently. More rarely march fracture, tenosynovitis of the Achilles tendon, chronic sprain of the knee, and low grade sciatica are found. A muscle hernia or hip disease can be the essential complaint. Meralgia paresthetica has not been infrequent as the true cause of symptoms. If any one of these conditions is present, that condition must be treated first.

Patients with Associated Peripheral Vascular Lesions. Associated peripheral vascular disease is a definite contraindication to surgical treatment of varicosities in the Army. These patients with intermittent claudication due to early Buerger's disease, Raynaud's disease, or arteriosclerosis, should be reclassified to limited duty or discharged from the service. While some authors[1] have rightly claimed that the peripheral arterial circulation is improved following saphenous ligation, the improvement is never marked enough to allow such a patient to return to full duty status.

Patients with Myalgic Asthenia. These are patients who, from heredity or environment, have insufficient strength and muscular development to withstand the arduous work of full-duty Army life. Often, they are men of over thirty-five years of age. Their habitus is such that they are unable to cope with the long marches and other physical labor demanded of a soldier, despite a careful conditioning program. They may not be psychoneurotic, and may have no physical illness, but they are as physically unable to compete with the average soldier as the ordinary well adult would be physically unable to compete with a first-class track man or a professional fighter. This type of patient rarely presents himself with the complaint that he is unable to keep up with his organization. He selects whatever physical disability is present and explains his inadequacy upon that basis. When this type of man has varicose veins he will naturally attempt to explain his difficulty upon the varicose veins. It is as much a mistake to operate upon these patients as it is to force them to perform full duty. They should be reclassified to limited service.

With these four groups eliminated in the consideration of each patient, the soldier suitable for surgery should have complaints referable to varicose veins alone and should be able to return to full duty following surgical treatment.

SIMPLE DIAGNOSTIC MANEUVERS

The primary pathology of varicose veins is an incompetent valve between the deep and superficial venous systems. These systems communicate directly by junctions between the long saphenous and common femoral, and between the short saphenous and popliteal. They are also connected by numerous perforating veins. Incompetent valves may occur at these two junctions or in any of the perforators. The purpose of any surgical procedure is the destruction of the open communication between the two systems by removal of the segment of vein containing the defective valve. Results obtained by surgery are directly dependent upon the preoperative localization of any and all incompetent valves.

Trendelenburg,[2] in 1891, first recognized the pathologic physiology and described the classical test to determine the state of the saphenofemoral valve. Since that time, and especially within the last ten years, when articles on varicose veins have appeared at the rate of one per day, innumerable tests and manipulations have been described for the determination of the site of the defective valve. These numerous

tests and the names attached to them have confused rather than simplified the diagnosis. The following routine avoids the use of "named tests," and gives an accurate diagnosis simply and rapidly.

The diagnosis of varicose veins is made by inspection, palpation, and manipulation. Inspection and palpation will locate the varicosities and help locate the defective valve. A bulge at the upper limit of a nest of varicosities is frequently the site of an incompetent perforator. The presence of a localized "nest" of varicosities may serve to bring attention to a particular branch of the great saphenous. After noting the distribution and severity of the varicosities, the answers to the following four questions determine the subsequent treatment: (1) Is the arterial supply adequate? (2) Is the deep venous circulation patent and adequate? (3) Is the saphenofemoral junction, or valve, competent? (4) Are there any incompetent perforators, and if so, where are they?

Is the Arterial Supply Adequate? Our test for arterial circulation is confined to palpation of the peripheral pulses, dorsalis pedis, posterior tibial and popliteal. A deficient arterial supply is a definite contraindication to surgery. There is some evidence to indicate that eradication of varicose veins improves a deficient arterial circulation.[3] However, in the military service such patients are not fit for full duty and treatment of varicosities is contraindicated.

Is the Deep Venous Circulation Patent and Adequate? To determine the patency of the deep venous circulation the extremity is wrapped from the toes to the groin in stockinette bandage cut on the bias (or Ace bandage). The patient is then allowed to walk. If the patient returns with the bandage in place and has no discomfort, the deep venous circulation is intact. When the deep circulation is not adequate, compression of the superficial veins or *collateral* circulation will produce pain, and surgery is contraindicated.[4]

Is the Saphenofemoral Junction, or Valve, Competent? A single tourniquet test determines the state of the saphenofemoral valve. The patient is told to lie down, his extremity elevated, and the veins emptied. A tourniquet is applied just below the fossa ovalis and the patient instructed to stand. Ordinarily, it will take forty to sixty seconds for the varicose veins to fill from behind through normal channels. If while the veins are empty, releasing the tourniquet causes the veins to fill immediately, the patient has an incompetent saphenofemoral junction or valve.

Are There Any Incompetent Perforators, and if so, Where Are They? Two tourniquets are employed to determine the presence and location of the incompetent perforator. After emptying the varicosities and applying the tourniquet just below the fossa ovalis, the patient is made to stand. If the varicosities fill within thirty-five seconds with the tourniquet in place, an incompetent perforator is present. This being the case, the tourniquet is released and with the patient in a supine position the varicosities are again emptied and the tourniquet applied. A second tourniquet is now applied about four inches below the first and the patient is again instructed to stand. If the veins again fill below the second tourniquet within thirty-five seconds, the procedure is repeated with the second tourniquet at a lower level. This procedure is repeated until the veins fill between the two tourniquets, indicating the perforator has been passed. By reapplying and gradually approximating the tourniquets and narrowing the field in which the veins fill, the exact site of the perforator is determined and marked. Not infrequently, additional incompetencies are present and are revealed by a filling of the veins below the second tourniquet. These additional perforators are located in a similar manner. In performing these tests, the examiner should bear in mind the high instances of perforators in the region about the knee, and the frequent incompetency of the short saphenous vein at its junction with the popliteal vein.

SURGICAL TECHNIC

Abundant evidence has accumulated to demonstrate the futility of injecting sclerosing agents into varicose veins in the presence of incompetent communications between the superficial and deep venous systems.[5,6] The pressure head upon the injected varicosities from the deep system either prevents thrombosis or results in early recanalization. Adequate treatment is based upon the surgical destruction of any and all open communications between the two systems by removal of the segment of vein containing the defective valve. Following this, and only then, is injection therapy indicated. Depending entirely upon the preoperative findings, a high ligation of the great saphenous, ligation of the short saphenous, ligation of an incompetent perforator, or a combined procedure is indicated.

Operative Technic. Anesthesia is obtained by skin infiltration of local procaine. A transverse incision is made parallel to and two-finger breadths below the inguinal ligament with its center one-finger breadth medial to the pulsation of the femoral artery. The incision is carried through the well developed superficial fascia using considerable care as the great saphenous lies immediately beneath. The great saphenous is isolated together with its constant tributaries, the superficial external pudendal, the superficial inferior epigastric and superficial external circumflex iliac veins. The saphenous and all its branches should be isolated and dissected free before any are severed.[7] The tragedy of ligating the femoral artery or vein is thus avoided. Dissection is carried distally for about 6 cm. and the vein doubly clamped and ligated. All branches are then individually clamped and ligated. After freeing the posterior surface of the proximal segment, double clamps are applied just below its entrance into the femoral vein. The vein is transfixed proximal to the first clamp with a needle and ligated. Another ligature is now tied just proximal to the first, thus sealing off any break in the intima of the vein wall. The segment is excised and closure is in layers. All buried sutures and ligatures are of chromic catgut No. 00. Interrupted silk is used for the skin.

If an incompetent perforator is present its location has already been marked before surgery. Through a transverse incision the vein is exposed and the communicating vein looked for. Here general traction upon the varicose vein will often cause the deep fascia to bulge around the point of perforation and will aid in localizing these perforating veins. The perforator is ligated as it comes through the deep fascia and the superficial vein is resected as much as is possible through the incision. Short saphenous ligations are done using a similar technic but the incision is made at the lower margin of the upper third of the leg posteriorly so as to catch the short saphenous as it passes through the crural fascia. Following the operation a dry dressing is placed over the wound and stockinette bandage is applied to the extremity reaching from the toes to the groin. This aids in the collapse of the veins and promotes thrombosis.

Postoperative Treatment. Walking is obligatory from the day of the operation. This reduces stasis and the danger of embolism.[8,9,10] The patient is usually discharged to full duty in about ten days to two weeks. He returns to the out-patient clinic the week following discharge where he is tested for sensitivity to sodium morrhuate by the injection of ½ cc. Following this, injections of 2 to 4 cc. are given at weekly intervals. A course of four to eight injections is generally sufficient.

Injection of sclerosing solution at the time of surgery is not done, despite its widespread use in many clinics. The advisability of such a procedure is questionable for the following reasons:[11] (1) The occasional severe reaction following this type of injection forces the patient to remain in bed. It is more important to keep the patient ambulatory than to hurry the sclerosing of the veins, for bed rest more than any other factor predisposes to

pulmonary embolism. (2) The few deaths reported following varicose vein surgery have been subsequent to injection of large amounts of sclerosing fluid at the time of ligation. (3) When perforating veins are excised, previous injection of the great saphenous may cause sclerosing solution to leak out into the lower wound and predispose to infection and sloughing of the wound. (4) To be effective the dose must be large. To avoid reaction from a large dose a test injection must be given first. This test dose may cause sufficient reaction to delay surgery and prolong hospitalization.

STATISTICAL SUMMARY

A total of 1,131 cases of varicose veins were seen as out-patients in the two years ending March, 1943. Of these only 340 were admitted to the hospital for study. Following more careful investigation 126 were eliminated as candidates for surgery, making a total of 917 rejected for surgery and 214 operated upon.

In the group of 214 patients operated upon, 206 had high saphenous ligation and of these 113 were bilateral; 106 cases had incompetent perforating veins, an incidence higher than that usually reported; seventy-five cases had one perforator and thirty-one had two, making a total of 137 perforators in the entire series. Distribution of the perforators was as follows: about the knee seventy-eight, short saphenous thirty-nine, upper leg twelve, and mid-thigh eight.

COMMENT

As can be seen, the criteria for selection of cases as outlined above was instrumental in eliminating 917 of 1,131 cases. For the proper treatment of Army personnel this is of major importance. Indiscriminate surgery in the rejected group would compound time loss, unnecessarily increase expense and aggravate postwar pension claims.

The simple diagnostic maneuvers described are sufficiently accurate to allow the localization of a high percentage of incompetent perforating veins.

The surgical technic involving high saphenous ligation is well established. The simultaneous excision of incompetent perforating veins is equally important. The fundamental pathologic physiology is the same in each case, for the saphenous vein as it enters the femoral is itself a communicating branch. It is as necessary to prevent the back pressure through incompetent perforators as it is to prevent the back pressure through the incompetent saphenous. While this principle has been recognized, some[3,7,8,9] have believed that satisfactory obliteration of the small incompetent perforators could be obtained by injection alone, either retrograde at the time of saphenous ligation or postoperatively. This has not proved to be true. The perforators are by no means small. Recurrences have appeared despite numerous postoperative injections in which perforators have been disregarded or missed, and have uniformly responded to subsequent ligation of such perforators. Since perforators are found in almost 50 per cent of the cases their adequate treatment cannot be minimized, and a definite plan for their obliteration must be included in any method of varicose vein management. Multiple incisions are no contraindication to their surgical excision. In less than 15 per cent of the cases were three incisions necessary, and in most two were sufficient. This method of combined ligation has in our experience prevented recurrence, shortened the period of treatment and lessened the total number of necessary postoperative injections. The average period of hospitalization has been 13.3 days, a relatively short period for patients who are returned to full duty.

Complications in this series were few. Two patients developed minor pulmonary infarctions following the development of postoperative thrombophlebitis in the operated leg. These were treated with repeated lumbar sympathetic blocks and

the usual supportive measures. There were seven minor wound infections in a total of 456 incisions and these occurred during a period when No. 2 chromic catgut was the only available suture material. There were no deaths.

SUMMARY AND CONCLUSIONS

1. Criteria for selection of cases suitable for surgery are presented for Army personnel with varicose veins.

2. Simple diagnostic maneuvers are described.

3. An outline of surgical technic emphasizes simultaneous high saphenous ligation and excision of incompetent perforating veins.

4. The results in 214 operations are given.

5. While the difficulties of follow-up in an Army Station Hospital make a long range evaluation of any series of cases impossible, the treatment outlined provides an adequate, effective, and safe method for the handling of varicose veins.

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TREATMENT OF BURNS*

SYMPOSIUM

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THE treatment of burns has undergone significant changes during the past two decades. Up to 1900, the treatment of burns was, more or less, a local treatment. Although laboratory studies were made and the significance of fluid loss and hemoconcentration (Baraduc, Tappeiner) and changes of the morphology of the blood picture (Wertheim, Lesser) were known, these important facts were, as a rule, not followed up therapeutically. It is estimated that two-thirds of the patients who were burned died from shock and infection. In the beginning of our century, the general treatment started to receive more consideration and was under the influence of the toxic theory. According to the latter, toxic substances are released from the burned area, causing shock and toxemia (Bardeen, Wilms, Pfeiffer, Dale and associates, Cannon and Bayliss, Robertson and Boyd). The toxic theory lost importance since Underhill and his associates' systematic investigation of the rôle of fluid loss in burns (1921–1923). Since this time, the treatment of burns has gone through four significant phases. Each phase has added a definite improvement, with a lowering of the mortality rate and the lessening of suffering of the patient.

In 1921, Underhill and his co-workers had the opportunity of treating twenty-one persons seriously burned in a theatre fire in New Haven. At the same time, they made important investigations of fluid loss and blood concentration following burns. Their investigations led them to the conclusion that the clinical picture of toxemia developing in cases of burns is not due to absorption of a toxic substance derived from burned areas but due to hemoconcentration. The blood capillaries became injured from the heat and became permeable; this results in a rapid pouring out of fluid on the burned surface and into the tissues, causing marked general edema of the affected part. The rapid and continued loss of fluid from the blood in cases of burns quickly induces a marked concentration of the blood. The condition of the patient depends on the amount of the concentration. Underhill considered restoration of blood concentration of prime significance. His systematic treatment of burns consisted simply in the forcing of fluid by mouth, rectum, hypodermoclysis or intravenous infusion. Restoration of normal blood concentration by adequate forcing of fluid was accompanied by marked evidences of improvement and the development of the usual sequels: delirium, unconsciousness, gastrointestinal disturbances, albuminuria, hemoglobinuria, of extensive burns were checked. Underhill and his coworkers later confirmed the clinical findings by animal experiments. They also were able to disprove experimentally any possibility of absorption of toxins from the burned area. Underhill's investigations were a remarkable step forward in the treatment of burns. For the first time, the important fact of fluid loss and hemoconcentration, although known before, was systematically followed therapeutically.

The next important phase in the treatment of burns was the introduction of tannic acid by Davidson in 1925. No other treatment up to that time had reduced suffering of the burned patient and lowered the mortality as much as the tanning

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A

B

Fig. 1. A, girl, four years of age with third degree burns treated with triple dye. She received 700 cc. of plasma in 445 cc. of 5 per cent glucose and 6 cc. of eschatine upon admission and during the next twenty-four hours. She received 250 additional cc. of plasma on the second day. Her hematocrit reading was 52 per cent cells on admission and dropped to 48.6 per cent within thirty-six hours. Sulfadiazine was administered and five blood transfusions given, 150 cc. each, within the next three weeks. The eschar started to come off three weeks after admission. The patient now received daily saline tub baths and sulfallantoin solution and saline dressing. The condition of the patient after the eschar came off is pictured in Figure 1B. The first skin grafting operation was performed four weeks after admission (Fig. 1C.) Axilla and surrounding areas were grafted first. Second operation two weeks later, (Fig. 1D.) The third and fourth operation two weeks apart from each other covered all raw surfaces. The patient was discharged four and one-half months after admission. Fig. 1E shows condition of patient six months later.

principle. According to the mortality statistics of the Bureau of Census, there were 8,083 deaths from burns in the United States in 1928, while there were 5,232 in 1933. This decrease has followed the introduction of the tannic acid treatment by Davidson in 1925–1927.* This treatment was based on the toxic theory. Davidson used tannic acid to precipitate any toxin that may be formed in the burned skin and to prevent it from becoming absorbed. It is a subject of considerable controversy, however, as to whether this theory is true. The present concensus of opinion is that the main benefit of the tanning principle lies in decreasing the loss of fluid from the burned area, relieving pain, preventing infection, and diminishing autolytic changes.

FIG. 1. For descriptive legend see page 35.

The third important phase in the treatment of burns started with the work of Aldrich and Firor (1929–1932). Their theory was that infection is the major cause for the toxemia in cases of burns. These authors were impressed with the fact that, as proved by positive cultures from the burned area and positive blood cultures, there is enough obvious infection present in all large superficial burns to account for all symptoms and physical signs exhibited by the patient. Their conception was that a burn is a large open

* Editorial. *J. A. M. A.*, May 30, 1942.

surgical lesion bathed in a virulent pus. Aldrich believed that the value of tannic acid lay in the formation of an eschar over a practically sterile burn, and the sealing of the burn which prevented fluid loss and contamination. In looking for an antiseptic substance to combat infection in cases of burns, Aldrich and Firor chose gentian-violet because of the bactericidal and bacteriostatic action of this dye. They also found that it not only was a specific against gram-positive organisms, but also had another beneficial quality: the dye reacted with the burned skin superficially to form a light, tough, flexible eschar. Furthermore, while tannic acid may damage the surviving epithelial islands, gentian-violet did not do it. The action of gentian-violet, however, is weak against the gram-negative organisms; therefore, Aldrich went one step further in devising a mixture of three aniline dyes: crystal-violet, brilliant green, and neutral acriflavine, known as triple dye, which is specific against the gram-positive organisms as well as against the gram-negative ones. Thus, Aldrich's and Firor's work pointed out for the first time the serious complications of infection in cases of burns. Since that time, various other treatments based on the infectious theory were devised, particularly since the dawn of the sulfonamide era. Sulfonamides administered by mouth have become almost a routine treatment in cases of burns. The local application has been advised by Pickrell and others. Pickrell sprays 3 per cent sulfadiazine in 8 per cent triethanolamine, which forms gradually an eschar over the burned area. It takes, however, four days with hourly spraying the first day, every two hours the second day, every three hours the third day, and every four hours the fourth day before an exchar is formed. Gower treats fresh burns with scarlet red bandages and moist sulfanilamide dressings. Cod liver oil as a dressing is also highly recommended by Lohr and others. Hardin, in reviewing the literature on that subject enumerates the following advantages: The oil is sterile and bactericidal; it is stimulating to granulation and epithelization; it accelerates liquefaction of the dead tissue; it produces a beneficial

Fig. 1e. For descriptive legend see page 35.

local and systemic response, and it acts also as a protective layer between the lesion and the dressing.

The fourth phase in the treatment of burns started with the introduction of plasma infusions. It was known before Underhill's and his co-worker's investigations that the fluid which escapes from the burned area was not merely water. Tappeiner (1881) pointed out that death from burns is due to hemoconcentration from loss of plasma and he was one of the first to advise the infusion of serous fluids. It was not until much later that his advice was appreciated, mainly through the investigations of Underhill and co-workers, Blalock, McIver, McClure, Harkins, Elman, Scudder, Moon, Lee, Elkinton, Wolff, Rhoads, Black and others. Normally, fluid is kept from leaking through the capillaries by the osmotic pressure of the

plasma proteins against the capillary blood pressure. If, as in burns, the capillaries become injured and permeable plasma colloidal properties. Whole blood transfusions are recommended less, since they may increase the cellular constituents of

A B

FIG. 2. A, contracture of right axilla from extensive burn. A strip of normal skin is visible between arm and chest. To correct the contracture, the binding scar is incised along the median border of the strip of normal skin; the contracture is reduced and the large defect resulting from reduction is covered with a thick split graft. B, same patient six months after operation.

escapes from the circulation, this results in reduction of the osmotic pressure within the capillaries. If, in such a state aqueous solutions are injected, the plasma proteins become diluted to a concentration that makes it impossible to hold the fluid given intravenously or by other means into the blood stream. In other words, in severe burns the more aqueous solutions are administered, the more fluid is poured out. What is needed is the replacement of the lost plasma. Weiner, Rowlette and Elman were among the first to stress the importance of replacing the lost plasma by plasma infusions, they also recommended the intravenous use of acacia due to its the blood that already has a high cell concentration. Elkinton, Wolff and Lee state that the capillary permeability with continued loss of plasma in burns lasts from thirty-one to forty hours. During this period, excessive hemoconcentration may be prevented by small repeated plasma transfusions. After the capillaries have regained their impermeability to protein, the deficit of plasma protein may be corrected quantitively by a large plasma transfusion. The amount of plasma required is calculated by a formula based on hematocrit values, plasma protein concentration and body weight. In order to decrease capillary permeability, or at

least to shorten its duration, the same authors recommend intravenous administration of adrenal cortical extracts, which have been used in the treatment of traumatic shock by Swingle and co-workers, Heuer, Andrus and others. The results of this procedure are, however, questionable. Thus, plasma transfusions have become a life-saving measure in severe cases of burns. As to the method of preparation of plasma, the work of Thompson, Ravdin, Rhoads and Frank; Strumia, Wagner and Monaghan; Scudder; Mahoney; Kingsley and Howland may be consulted.

CLASSIFICATION OF BURNS

Thermal burns are usually classified according to penetration.

The first degree burn, combustio erythematosa, is characterized by an erythema of the skin from enlargement of the dermal vessels due to paralysis of their nerves.

The second degree burn, combustio bullosa, is characterized by formation of blisters between the epidermis and the corium.

In third degree burns, combustio escharotica, the entire epidermis is destroyed. The eschar is hard and insensitive, brownish or black; the vessels are thrombosed, the surrounding parts exhibit burns of first and second degree.

CLINICAL PICTURE

The clinical picture of a burn depends mainly upon the extent of the involved area. Minor burns cause only local reaction: extensive burns, however, cause general reaction which may run through the following stages: primary shock, secondary shock, acute toxemia, septic toxemia, healing or death.

Shock. Shock, in general, is divided into primary and secondary shock. Primary shock is the collapse which follows the trauma immediately, and is probably due to pain and to various other reflexes and psychological factors; it is usually not of

A

B

FIG. 3. A, contracture of right axilla and elbow joint after third degree burn of right arm, chest, abdomen and back. A tube flap was prepared for correction of the extensive contracture of the elbow joint from the left thoraco-epigastric region (the right side was scarred). B, the flap was transplanted using the left wrist as a carrier. Contracture of right axilla was corrected with "z-operations." Note length of flap on flexor region of elbow joint two years after the operation.

serious consequence. Secondary or traumatic shock is the circulatory collapse that follows one hour after the injury and is characterized by pallor, cyanosis of lips, cheeks, and extremities, cold, moist, sweaty skin, rapid, thready pulse, rapid, shallow respiration, restlessness, nausea, vomiting, falling blood pressure, subnormal temperature, increased hematocrit and leucocytosis. In cases of burns, as in any other traumatic cases, primary and secondary shock may merge imperceptibly into each other.

Toxemia. The well known syndrome of toxemia follows extensive burns within the first three or four days, if the patient survives the secondary shock. There is no agreement as to the nature of toxemia. The fact that it can occur or persist after the capillaries have regained their impermeability would exclude hemoconcentration as the sole cause; absorption of toxic substances and infection may also play a rôle. Vomiting is the first evidence of beginning toxemia. It is soon followed by a typical syndrome which is characterized by mental and physical disturbances. The patient becomes restless, complains of thirst and pain, at times is delirious; he is unable to retain food, temperature rises and remains high, and the condition soon assumes a septic character. Pulse and respiration are frequent. The tongue is dry; the blood pressure may remain normal. The leucocytes are markedly increased (25,000 and more). The urine is scanty and concentrated, albumin and acetone may appear. Hemoconcentration with a rapid increase in the hematocrit readings, although present early, may reach its peak at the onset of the toxic stage. Blood chlorides and plasma proteins are reduced, blood sugar and nitrogen elevated. This picture of toxemia may last from one to two weeks. Then, if the patient survives, the patient's mental and most of the physical disturbances clear up. However, there may remain an elevated temperature and pulse, marked leucocytosis, and evidence of secondary anemia and hypoproteinemia until the greater part of the raw surfaces have healed.

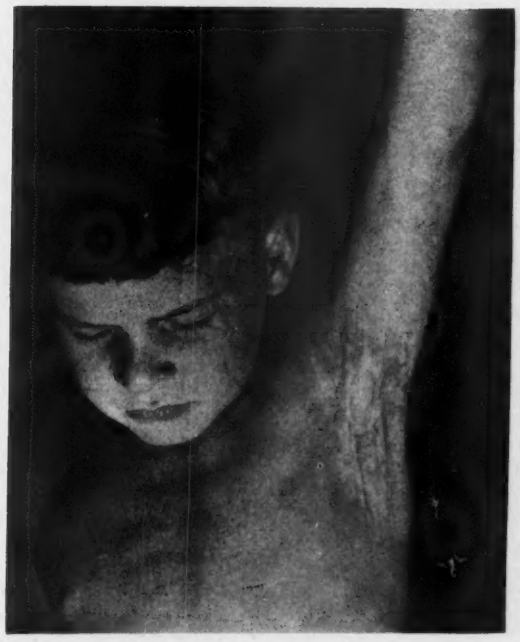
Infection, as already mentioned, is partly responsible for the toxemia syndrome. But, it may recur at a later date, days or weeks after the symptoms of acute toxemia have disappeared. The symptoms and findings of the septic toxemia (Wilson) resemble those of any other surgical septicemia. The prognosis is grave; fatal signs are apathy and semi-consciousness. Blood pressure drops, pulse becomes thready, respiration accelerated and shallow, vomiting, diarrhea, clonicity, coma, collapse, and a marked rise in temperature precede the fatal issue.

TREATMENT OF BURNS

Treatment of Extensive Burns. The treatment of extensive burns can be divided into an immediate and late treatment. The immediate treatment consists of combatting shock, toxemia and infection. The late treatment consists of covering of the raw surfaces and correction of contractures.

Immediate Treatment. Almost every patient with extensive burns exhibits more or less evidence of shock, which must be treated first before proceeding with any local treatment. Everything should be done to combat shock and everything should be avoided that would increase it. Pain is relieved with an injection of morphine. The clothes are removed with the least disturbance to the patient. The burned area is wrapped in sterile sheets. The foot of the stretcher or bed is elevated; the patient is wrapped in warm blankets, heaters, such as hot water bottles, etc., are placed around him. Inhalation of oxygen may be of value. While this is being carried out, the most important measure to combat shock is prepared and administered: restoration of blood volume by continuous intravenous infusion of plasma and 5 per cent dextrose in distilled water. If plasma is not available, transfusion of whole or citrated blood may be substituted, or gum acacia or dextrose alone. Of drugs

A



B

FIG. 4. A, contracture of left axilla. The contracture was due to three bridle scars which were broken up by "z-operations." The "z" of the anterior web has been outlined in Figure 4A. B, six months after operation.

to combat shock, only adrenal cortical extract, known as Eschatin, (5 to 10 cc. injected intravenously every six hours, or Percortin, 2 to 4 cc. injected intramuscularly every four hours) may be of value. Even in those cases which do not exhibit evidence of severe shock, plasma transfusions and administration of adrenal cortical extracts should be given for the purpose of combatting fluid shifting and hemoconcentration. Only if there is evidence that shock is controlled, may local treatment be instituted.

Any burned area must be considered as a surface wound and treated as such. Rigid asepsis must be carried out. The patient is placed on an operating table which is covered with a sterile sheet. The operating room personnel is prepared as for any aseptic operation. After an injection of morphin or under light anesthesia if necessary the cleansing and débridement is done as follows: Oil or grease, if it had been used as a first aid treatment, is removed with ether or benzene. The entire burned area is gently washed with soap and water and rinsed with saline solution. The area is now draped with sterile sheets, all blisters are opened, and the loose, necrotic skin is removed. Then the entire area is flushed with copious amounts of warm normal saline solution and dried, if possible, with a hair dryer. The entire burned area is treated with the "sealing" methods or by open drainage. Since the introduction of plasma infusions to combat fluid loss the "sealing" methods have lost popularity.

Tannic Acid Treatment. Tannic acid silver nitrate, as recommended by Bettman, has several advantages as contrasted with tannic acid alone. The main advantage is the fact that coagulation can be accomplished in minutes, while hours are necessary if tannic acid alone is used. A freshly made 5 per cent tannic acid solution is applied with cotton swabs to the burned area. The surface dries quickly. Following this, a 10 per cent silver nitrate solution is applied in the same manner, causing immediate coagulation. This procedure is repeated until a satisfactory eschar is obtained. After twenty-four hours, freshly formed blisters are opened and treated in the same manner. Tannic acid treatment when correctly carried out according to rules laid down by Davidson himself and such men as Penberthy and McClure, who were closely associated with him, and Bettman, gives good results. Serious arguments have, however, arisen recently against its use, of which the destructive nature of the tannic acid against surviving epithelial islands (Cannon and Cope, Hirschfeld) and the inflexibility of the eschar are the most important ones.

Triple Dye Method. The formula of triple dye is crystal violet 1 per cent, brilliant green, ½ per cent, neutral acriflavine ¼ per cent dissolved in distilled water. The triple dye is sprayed over the dried, burned surface. The eschar is now dried again with a hair dryer, followed by another layer of triple dye, which is dried again. Usually, by that time, a thin, flexible eschar is produced and analgesia is obtained. Any blisters developing later on are treated in the same way.

Vaseline Gauze and Compression Dressing Method (Brown, Koch, Mason, Allen, Siler and Reid, Cope, Cannon). With the injured surface cleansed as completely as possible it is smoothly covered with a few layers of fine meshed gauze impregnated with petrolatum. Such gauze is easily prepared in strips three inches wide and twelve inches long and sterilized in a porcelain basin provided with a well fitting cover. This dressing does not adhere to the raw surface as does a coagulant crust; it does not fix tissue and it still permits drainage of serum and exudate into the dressings outside it. Koch's instructions are:

"Over the petrolatum gauze is laid a half dozen layers of flat, dry, sterile gauze, over this a mass of gauze fluff and over the fluff, mechanic's waste or sea sponges so as to provide under the retaining bandage a resilient covering that produces an even

pressure over the injured extremity or the burned surface but does not cause constriction.

"The dressing and compression sponges are held in place preferably with an elastic bandage such as stockinet. This is particularly advantageous if the burn involves the trunk."

Burned limbs are immobilized with splints. This primary dressing is changed after twelve to fifteen days and then at regular intervals of twenty-four or forty-eight hours with removal of any adherent tissue that can be cut away without causing pain or bleeding. This treatment pursues the same purpose as the tanning principle: covering a large, open wound which has been changed from a contaminated to a clean wound with an occlusive dressing to check the danger of infection; secondly, checking consistent loss of serum from the burned area and into the tissue by pressure dressings.

The advantage of this treatment as contrasted with the tanning principle lies in its quicker and simpler application, open drainage and quicker preparation of the area for skin grafting.

After the application of the local treatment, the patient is placed under a thermoregulated heat cradle; the intravenous infusion is kept running at a speed of about 40 to 60 drops per minute. Hemoconcentration and plasma protein levels are determined every six hours. The amount of plasma administered intravenously depends upon the degree of the hemoconcentration. The desired amount is calculated according to formulas, such as recommended by Lee and his co-workers, (Wolff, Elkinton, Rhoads), who based the treatment of burn shock upon a quantitative basis. This formula takes the body weight, the plasma protein level and the hematocrit level into account. A simple but not as accurate method of calculating plasma dosage in burns was devised by Harkins: Give 100 cc. of plasma for every point the hematocrit is above the normal of 45. This formula does not take the existing plasma protein level into account. Plasma is administered until the hematocrit value remains around 50 per cent cells and the plasma protein level around 6 Gm. per cent. When laboratory facilities are not available, the amount of plasma to be given is calculated according to the extent of the burned body surface (Harkins). According to Berkow's evaluation of surface area of the body, the percentage of the burned area is estimated—head-neck 6 per cent, upper extremities 18 per cent, trunk 38 per cent, lower extremities 58 per cent—and 50 cc. of plasma should be given for every per cent of the body surface affected by a deep burn. The formula roughly estimates the entire amount of plasma that will be necessary; one-third of this amount should be administered during the first two hours; one-third the next four hours, and one-third the next six hours.

Sulfanilamide orally or intravenously administered is strongly recommended; the oral administration may be impossible due to vomiting.

Further treatment consists of improving the patient's general condition and if the tanning principle has been used careful watching of the eschar for any evidence of infection. If tannic acid is used, it may be difficult to locate the infected area. Drilling of the eschar in various places may be necessary. If the infected area is found, the eschar must be dissected away. If triple dye is used, the eschar becomes soft and moist over the infected area and can easily be removed with scissors.

If no infection intervenes, the eschar is allowed to stay until it comes off by itself. In second degree burns, the coagulum peels off in several weeks, leaving an epithelialized red, dry, skin surface beneath. This process may be speeded up by application of compresses soaked in hypertonic saline solutions. Hypotonic saline solutions are not recommended, since they may lead to absorption of autolytic material and toxic symptoms. In third degree burns, the coagulum peels off in some places, leaving

a granulating surface behind, and remains in other places. The remaining eschar is covered with hypertonic solution. Daily baths in hypertonic solution are recommended. If the open drainage principle is used, the sloughing areas are débrided on changing each dressing.

Late Treatment. Whenever indicated, skin grafting should be done as soon as possible. If a granulating surface is left—as in every third degree burn—it should be skin grafted. The quicker epithelialization is brought about, the less will be the degree of contracture. Brown and McDowell, in a recent monograph, give an excellent description of the healing process of the burned surface. However, it must be remembered that skin grafts will take only on pinkish, flat, healthy looking granulations. If the granulating area is chronically infected and the granulations are sluggish, hypertrophic, and gray, the healing process should be stimulated by general and local measures before skin grafting can be successful. Frequent blood transfusions until the hemoglobin is well above 55 per cent, administration of vitamins and iron in a high caloric diet are paramount. Daily saline baths, exposure to sunlight or ultraviolet rays stimulate not only the local but also the general condition. The granulating area may be treated with local application of sulfonamide drugs (not more than 15 Gm. daily), together with wet dressings such as hypertonic saline solutions, Dakin's solution, or sulfallantoin, (Koerber and associates and others). I found the latter solution particularly valuable in hastening liquefaction of dead tissue and stimulating the growth of healthy granulations. Or ointments may be used such as cod liver oil ointments, scarlet red, etc. Application of zinc peroxide has been recommended for those granulations which tend to bleed (Altemeier and Carter). Most important is the application of a pressure dressing to counteract edema and to prevent the granulations from becoming edematous. If, in spite of all measurements, the granulations remain edematous and boggy, skin grafting should be started nevertheless. The host area should be sprinkled with sulfanilamide, which does not interfere with the "take" of the graft (Converse). If, in spite of these precautions the grafts do not take, thin split grafts about the size of a thumbnail—so-called "island grafts"—should be used, or seed grafting may be attempted. However, if granulations are pinkish and healthy, the transplantation of a large, thick, split graft is the preferred type. Those regions which tend to contract, such as the flexor surfaces of the extremities or the junctions of limbs and trunk should be covered first. A several-stage procedure may be necessary; the same donor areas may be used several times. As many as four times, grafts have been taken from the same area. If contractures have already developed, they should be corrected as soon as possible (May).

Treatment of Extensive Burns of Face, Hands, Feet, Perineum and Genitalia. These areas deserve special consideration. No coagulant should be used for burns in these regions; a coagulant on the fact, particularly around the eyes and mouth, is uncomfortable for the patient; at hands and feet it may constrict circulation, and at the perineum and genitalia, it harbors infection. After thorough débridement, the burned surfaces are covered with an ointment such as sulfanilamide ointment in combination with cod liver oil or Foille, which due to its analgesic effects is recommendable, (Hamilton, C. B. Owings, Personal Communication) or boric ointment. The face is then covered with fine meshed gauze. Deep burns of the eyelids tend to contract the eyelids and to evert them, causing an ectropion; suturing the eyelids together may counteract contraction.

The fingers and toes are best treated individually to prevent adhesions. Each finger is wrapped in fine meshed gauze in which the ointment is incorporated. The entire hand is then wrapped in surgical gauze; the hand, including the arm, is

placed on a padded splint and immobilized; the splint is kept elevated. The dressings are changed every two days. They are soaked off in saline baths. In third degree burns, early skin grafting is necessary.

Perineum and genitalia may be treated with ointment and covered with mesh gauze. The vulva, however, and anal regions are best treated with gauze soaked in antiseptic solution such as merthiolate and others. Frequent changings are necessary to prevent infection and avoid adherence of the gauze to the burned area.

Treatment of Minor Burns. Minor burns may be called burns of less than 5 to 10 per cent body surface. In most instances, they can be treated ambulatory, unless the location of the burned area is such as to require hospitalization. They usually do not require any general treatment, but the relief of pain by administration of a sedative. In first degree burns, application of antiseptic and analgesic ointments, such as Foille, or butesine picrate is recommended. The latter is an excellent analgesic, but should be used with caution, i.e., not repeatedly, since it may cause toxic symptoms. Second and third degree burns require débridement and the application either of an antiseptic analgesic ointment, or application of a coagulant. If the latter is used, triple dye is preferred for the flexible properties of its eschar. The burned area should be protected with sterile dressings; application of splints may be necessary; ultimate treatment does not differ from that of extensive burns.

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SKIN AS A SOURCE OF SYSTEMIC INFECTION

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SEPSIS signifies that bacteria enter the blood stream, form a focus in the body and give rise to local and systemic symptoms (Schottmueller). For the successful treatment of sepsis the location of the primary focus is of the utmost importance. In this communication the skin as a primary focus of systemic infection will be discussed.

Since the advent of sulfachemotherapy, the mortality in cases of sepsis has been immensely reduced. Chemotherapy, however, does not eliminate the necessity of locating the primary focus because in many instances the removal of the primary focus is actually life-saving and necessitates a minimum of chemotherapeutic application. In other instances the eradication of the primary focus prevents the establishment of a secondary focus which either immediately or remotely, may cause serious sequellae.

In order that the skin may serve as a portal of entry, there must be an interruption in its continuity. The skin, particularly the exposed parts, is subject to injury, which frequently occurs among workers who handle sharp tools in such occupations as meat-cutting, skinning, stone-cutting. Ordinary skin conditions such as acne vulgaris, dermatophytosis or impetigo, may occasionally harbor virulent bacteria, which in turn may enter the blood stream and cause sepsis.

It is rather remarkable that a small furuncle or carbuncle may completely heal and leave in its trail bacteria in some distant tissue of the body, which later may constitute a secondary focus of infection.

One may wonder why the skin is not the cause of general sepsis more frequently. The outstanding reason is the strong specific tissue immunity of the skin. This has been demonstrated by innumerable experiments. Clinical appreciation of this fact is substantiated by the following observations. Most infectious and contagious diseases characterized by exanthema endow the afflicted individual with permanent immunity. Thus, in scarlet fever, measles, small pox, typhoid and typhus, there is no recurrence after recovery. On the other hand, in infectious or contagious diseases without skin manifestations, such as diphtheria or pneumonia, recurrences are not rare. Cases of syphilis without exanthema of the skin in the secondary stage are more likely to develop tertiary cerebrospinal or vascular involvement, which explains the unawareness of a primary syphilitic infection in many patients who present themselves with cerebrospinal or vascular lues. The physician usually states that the patient denied lues. The patient is actually ignorant of the fact that he had a primary infection because he failed to show any skin manifestations which might have caused him to seek treatment, and thereby prevent the hazardous tertiary incurable lesions. Further evidence of the importance of the skin in serving as a protective mechanism against infection, are the local Schwartzmann phenomenon, and the fact that both sensitization and desensitization of disease are exercised through the medium of the skin.

However, local tissue immunity may at times be unable to resist the extraordinary virulence of the invading organism, so that a small interruption of continuity of the skin may be followed by serious systemic infection almost immediately.

There are two groups of bacteria which cause systemic infection. The septicemia group is caused most often by staphylococci, less often by streptococci, and occasionally by the gas gangrene group of organisms. The toxin producing organisms are represented mainly by Clostridium tetanus and occasionally Bacillus anthrax.

STAPHYLOCOCCIC SEPSIS

Staphylococcic sepsis, according to clinical manifestations, may be divided into five groups:

1. *Slight or No Visible Injury of Skin with Fulminating Fatal Sepsis. A Reactive Type.* The patient has severe chills, hyperpyrexia, and even delirium, very frequently positive blood culture of Staphylococcus aureus hemolyticus. Most often leucopenia and a marked shift to the left is present. Not infrequently immature white cells and large numbers of myelocytes are found in such abundance as to make it impossible to differentiate it from aleukemic leukemia, except by a study of the bone marrow. The tongue is dry and parched. Death often follows in forty-eight hours. The physical examination is almost entirely negative. Postmortem examination shows cloudy swelling of the liver, heart and kidneys, and most of the capillaries are filled with thrombi. This form of sepsis has been termed cryptogenetic because the focus of infection cannot be located. It is due to the fact that there was complete lack of immunity.

2. *Demonstrable Local Lesion, Removal of Local Lesion Preventing Fatal Sepsis.* This can best be illustrated by the following case:

A girl of nineteen suddenly developed chills and very high temperature (in the era preceding the use of sulfa drugs). On examination, the only evidence of infection was a slight discoloration of the little finger. Roentgen examination of the phalanges was negative. However, because of the local infection and violence of systemic symptoms, amputation of the terminal phalanx was deemed essential. At operation an infected thrombus was found in the vein which was ligated. Postoperatively, for one or two hours, the patient had chills and marked elevation of temperature. For two days, blood cultures showed a number of colonies of Staphylococcus aureus hemolyticus. On the third day, the temperature subsided, and within four or five days after operation the patient recovered.

Certain areas of the skin, namely, neck, lips, nose and eyelids, when infected give rise to sepsis more often than other parts of the body. This is due to the fact that in these regions more direct communications with the veins exist such as the maxillary veins, the occipital and ophthalmic veins and the cavernous sinuses. When an infected thrombus lodges in one of these veins, severe systemic symptoms will result, frequently with fatal termination. However, in this era of effective chemotherapy with sulfa drugs or with the promising penicillin and gramacidin, those lesions inaccessible to surgical intervention may be remedied.

3. *Local Focus of Skin with Minimal Local Symptoms Causing Invasion of Lungs.* Staphylococcus aureus septicemia may manifest itself as a systemic infection with multiple metastatic purulent foci, although the primary focus is not discernible.

Such a case was that of a physician, age forty-five years, who complained of pain in the throat, with high temperature but no throat exudate. On the second day he developed diffuse abdominal pain. Physical examination revealed marked tenderness over McBurney's point, but no rigidity or rebound tenderness. The leucocyte count was high, with 85 per cent polymorphonuclears. It was suspected that the patient had an acute appendicitis, and he was removed to the hospital. However, operation was delayed because of the absence of rigidity and muscle spasm, and the high temperature. After twelve hours there were definite signs of pneumonia over the right base. Blood culture showed, within twenty-four hours, a large number of colonies of Staphylococcus aureus hemolyticus. The diagnosis was evidently staphylococcemia with a metastatic focus in the lungs, and was later confirmed by

the expectoration of large quantities of pus containing Staphylococcus aureus, and by the presence in the chest of encapsulated areas of cloudy effusion. The aspirated fluid likewise contained Staphylococcus aureus. The entire course of the disease, which was three months, was characterized by continuous high temperature and severe thoracic pain. Improvement was gradual and eventual recovery complete. Treatment was symptomatic, aspiration of the purulent fluid and numerous blood transfusions, since sulfachemotherapy was not yet known. It was subsequently disclosed that several weeks before the acute illness, he had an infection of the toe with moderate lymphangitis of the leg and swollen glands in the groin.

4. *Secondary Invasion of Endocardium.* Secondary invasion of the endocardium is one of the most serious complications of staphylococcic infection of the skin. If the septic endocarditis is acute, fatal termination occurs within five days, at most within two weeks. In cases in which the staphylococcic endocarditis is subacute, the condition is likewise fatal, but the patient may linger for eight to ten months before succumbing. In all cases of subacute endocarditis there must have been a previously diseased valve.

An illustrative case is that of a young man in the midtwenties, who had a chronic rheumatic valvular lesion and diffuse eczema of the skin. When admitted to Beth Isreal Hospital, he had moderate elevation of temperature, slight anemia, no enlargement of the spleen and no petechiae. Because of the skin lesions subacute bacterial endocarditis was suspected. After several blood cultures, a few colonies of Staphylococcus albus were recovered,—a finding thought to be due to contamination. With the administration of sulfathiazole because of persistently elevated temperature, the blood culture became negative. Three weeks later, when the temperature was normal and the blood cultures had been continuously negative, the patient was about to be discharged from the hospital under the impression that the sulfathiazole had had a curative effect, or that the staphylococcus had actually been a contaminant. Two days prior to the supposed date of discharge, blood culture was again repeated, and a large number of Staphylococcus aureus hemolyticus colonies were recovered. Despite the continued use of sulfathiazole the patient developed multiple petechiae, embolic manifestations of the spleen, and succumbed two months later. Autopsy was not obtained.

Another example of metastatic foci arising from a non-discernible primary focus, is that of a physician, aged thirty-eight years, who developed chills, high temperature and vague abdominal pains, cough without expectoration, and physical signs of bronchopneumonia. Staphylococcus aureus hemolyticus was found on blood culture. At the end of four weeks he developed staphylococcic endocarditis, and succumbed a few days later. Postmortem examination showed multiple abscesses of the lungs, kidney, brain and heart, with ulceration of the mitral valve. The primary focus could not be determined.

In most cases of this type it is reasonable to assume that some minor skin infection, such as dermatophytosis, can be the initial lesion responsible for the virulent infection. We have encountered one case with the primary focus of infection in the external meatus of the ear, and another in the peri-anal region.

5. *Sepsis from a Reactivated Dormant Focus.* The bacteria enter the blood stream from the primary focus in the skin and are deposited in some distant part of the body where they may give rise to infection, constituting a secondary focus. The most common examples are staphylococcic carbuncle of the kidney, and para- or perinephritic suppuration occurring weeks or months after a minor staphylococcic infection of the skin. The staphylococcus has a specific predilection for the cortex of the kidney; the more severe the septic infection, the greater the renal damage. Hence multiple miliary abscesses of the kidney are usually found which are the immediate responsible factors for fatal termination. If the staphylococcic infection of the skin is mild in its local expression, only superficial lesions of the kidney may result, constituting a locus minoris resistancia ready to flare up when the individual is debilitated. It should be emphasized that whenever a patient develops chills

and elevation of temperature plus moderate anemia, vague abdominal symptoms and tenderness over a loin, even to a slight degree, thought should be given to the possibility of a carbuncle of the kidney resulting from a hematogenous infection where the primary source has been a minor skin lesion. *X*-ray examination may aid in the differential diagnosis. Through the independent studies of the late Edwin Beer,[1] and of Lipsett,[2] flat films of the abdomen with special attention to the region of the kidney may give significant early information. Both authors found in peri- and paranephritic infection, obliteration of the psoas muscle and scoliosis of the spine with the concavity toward the affected side. However, we have encountered two cases in whom these *x*-ray signs were conspicuously absent, causing undue delay in surgical intervention. Such experiences must be borne in mind. In peri- or paranephritic infection and carbuncle of the kidney, the blood culture is almost invariably negative.

There is a group of cases occurring in children or young adults in which the staphylococcus has gained entrance through the skin and the organism deposited in the bones. The bacteria have remained dormant until trauma, even of a minor degree, serves as an exciting cause in producing osteomyelitis. Such patients may have an onset with severe systemic symptoms, chills, high fever, delirium, and local signs of tenderness only on deep pressure. At this time *x*-rays are entirely negative. Within twenty-four hours, the local swelling increases markedly and the pain becomes excruciating in character.

Because we are fortunate to have the use of sulfa drugs, active chemotherapy should be instituted even before local signs make the diagnosis obvious.

In these cases, blood cultures are invariably positive. Of great clinical importance is the presence of staphylococcic cervical osteomyelitis of one or more vertebrae from the third cervical, down. A minor exciting cause may activate the dormant bacteria and give rise to symptoms. The onset is sudden, with high temperature, chills, severe occipital headaches radiating down the spine. There is marked local tenderness over the cervical vertebrae. This condition is erroneously diagnosed as meningitis. However, lumbar puncture is always negative. *X*-ray examination does not as a rule reveal the lesion. But if the clinical picture is borne in mind early surgical intervention is the only means of saving life. A delay of a few days will result in fatal perimeningeal abscess, or invasion of the cervical cord, with resulting bulbar death.

STREPTOCOCCIC INFECTIONS

Streptococcic infection originating in the skin is rare. It is usually secondary to a primary focus in a mucous membrane, or in a viscus. However, in cases of streptococcic infection originating in an interruption in the continuity of the skin, the local reaction is sufficiently violent to cause severe local infection and suppuration, so that systemic invasion can be checked by proper treatment.

Several years ago we saw a case at the Beth Israel Hospital, of a twenty-seven year old woman who was admitted with evidence of septicemia, Streptococcus hemolyticus having been recovered in the blood culture. The primary focus was an infection around the umbilicus. The patient was not mentally fit to give a history of the origin of the infection, but it is most likely that the streptococci entered the blood stream through this primary infection. Postmortem showed streptococcic endocarditis and miliary abscesses of the heart and kidneys.

Streptococcic skin infections can give rise to rapidly progressive septicemia from an infected hair follicle of the skin. Also, streptococcic furuncles or carbuncles especially on the neck, may be a primary focus for septicemia. A common source of streptococcic sepsis through the skin is seen after punctured wounds acquired during surgical operations by the surgeon, and by the pathologist when performing

an autopsy. Such infections may give rise to severe local reaction, and sometimes to multiple abscesses of the kidney, myocardium and septic endocarditis. In streptococcic skin infections it is not uncommon for multiple metastatic abscesses to occur in the muscles of the abdomen, gluteal muscles or gastrocnemius. In contradistinction to staphylococcic sepsis, lung abscesses are extremely rare in streptococcic sepsis.

Treatment. With the advent of the present chemotherapy, the prognosis in the non-fulminating cases of sepsis has become very much better. It may be roughly stated that both in staphylococcic and streptococcic sepsis, the mortality has dropped from 65 to 100 per cent, to between 8 and 10 per cent.

There must be early and vigorous administration of the sulfa drugs. The initial dose, if the patient does not vomit, should be 2 to 3 Gm. orally and the same dose to be repeated in four hours. This should be followed by 1 Gm. every four hours, daily, until four days after the symptoms have abated. The usual precautions should be taken of examination of the blood and the urine. Crystal formation in the kidneys can be minimized by the intake of alkalies and large draughts of water.

Most observers have found that sulfathiazole acts best bacteriostatically on the staphylococcus, whereas sulfapyridine, sulfanilimide and sulfadiazine act best on the streptococci. If the patient does not respond sufficiently to the particular sulfa drug that is being given within a period of twenty-four hours, the drug should be replaced by one of the other sulfa preparations.

Sometimes, where we desire a fast high blood concentration, or if there is vomiting, intravenous administration of the sodium salts of sulfathiazole or sulfadiazine should be given.

Despite the efficacy of sulfa therapy, a suppurating focus, if accessible, must be surgically eradicated.

In severe cases it is necessary to maintain the fluid and electrolytic balance by intravenous administration of saline and glucose. Blood transfusions should be given when indicated.

Sera, vaccines, toxoids and bacteriophage, which at best were of very doubtful value, are at present in the background since the use of sulfa drugs.

Particularly for staphylococcic sepsis, penicillin promises to be a useful drug.

TETANUS

Because of the war, this disease must be discussed in greater detail. Tetanus is caused by a spore-forming anaerobic organism introduced into traumatized skin. It has been experimentally demonstrated that tetanus spores injected in tissues under aseptic conditions do not grow. Recent experiences in the present conflict have shown slight wounds, that were directly covered with gray wool for padding purposes, in some instances became infected with tetanus. Other sources of infection have been contaminated catgut and intramuscular injections. As is well known, tetanus organisms are present in the soil, and a common inhabitant of the intestines of domestic animals, particularly horses.

One of the most commonly accepted theories today is that tetanus toxin is fixed by the central nervous system tissues. This fixed toxin then becomes altered, and the altered toxin causes death by respiratory paralysis. It is, therefore, necessary to institute treatment before a lethal dose of the toxin becomes fixed by the tissues of the central nervous system. From this it follows that with the detection of the earliest possible sign of the disease, treatment must be vigorously instituted.

The most common early symptom is trismus with pain in the angle of the jaw and muscles of the neck. This progresses to the familiar facial expression known as risus sardonicus. Within twenty-four hours in the severe cases, and in the milder cases between forty-eight hours and four to five days, reflex spasms set in. This consists of

a sudden spasm of the jaws and trunk, into tonic contraction. The back and chest become arched (opisthotonus), or the trunk may be bent forward (emprosthotonus), or laterally (pleurosthotonus). The patient may die in one of these reflex spasms due to respiratory failure. In severe cases, death usually occurs within five days. Survival beyond this is in favor of recovery. Those who recover are rarely left with sequelae.

Treatment. When the diagnosis has been established, 50,000 units of antitetanic serum should be immediately injected intravenously. It is Firor's[3] contention that this amount can adequately counteract any sublethal dose of tetanus toxin. If a lethal dose is present, any treatment is useless. Since the symptoms in a patient can be just as severe with a sublethal as well as a lethal dose, it is not justifiable to withhold prompt and adequate treatment.

At the time of the intravenous injection, the area about the infection should be injected with 10,000 units of antitetanic serum, in preparation for wide excision of the wound. One should wait an hour before operative treatment, so that the area around the wound is sufficiently saturated to neutralize the toxins in the operative area.

Fifteen thousand to 20,000 units of serum can be given intrathecally or intracisternally. This route has been objected to recently because of experimental observations. It has been shown that the introduction of serum into these areas does not directly neutralize the toxins in the tissues of the central nervous system. The only route to these tissues is through the choroid plexus which can be reached only through the blood stream. The more direct route, therefore, would be the introduction of antitetanic serum intravenously. In addition, the intrathecal or intracisternal administration of serum may cause serous meningitis that may aggravate the symptoms.

For the control of reflex spasms 5 Gm. of chloral hydrate per rectum may be used. Paraldyhyde 10 cc. to 40 cc. every three hours may be tried. Avertin has been found efficacious in dosage of 60 to 80 mg. per kilo of body weight. This controls the spasms for about six hours. It may be repeated a great many times, if necessary, with safety.

Since the introduction of prophylactic immunization, the disease has become quite rare. Passive immunization has resulted in a more prolonged period of incubation, a greater number of cases showing only local tetanus, and a marked reduction in mortality. Passive immunization is effected by giving an intramuscular dose of 3,000 units. This can be repeated in ten days.

At the present time, active immunization is practised. Three doses of alum precipitated toxoid may be used. The initial dose is 1 cc. subcutaneously. A similar amount is given one to three months later and a third similar dose one to three months after the second dose. Different time intervals are being tried out by the armed forces. Recent reports of active immunization against tetanus in this war show excellent results.

GAS GANGRENE

This condition is caused by gas producing organisms in wounded tissues, particularly muscles. The most common offending organism is the Clostridium welchii, with Clostridium septique and oedmatiens next in order of frequency.

It is true that the gas gangrene producing organisms are inhabitants of the soil, and that contaminated soil was the direct source of infection in these cases. However, recent observations, both clinical and experimental, have demonstrated that the infection is carried into the wound by the wounded wearing woolen clothing. All domestic animals harbor anaerobic organisms, but the sheep is the most important source of infection because its wool is used for clothing. Maes[4] did the following experiments to substantiate wool as the

source of anaerobic infection. He took twelve samples of wool, cotton and silk, dry-cleaned and pressed half of each sample. Cultures were then taken from the cleaned and unprepared portions of the samples. In eleven out of twelve pieces of wool, anaerobic organisms were found. None were found in the silk or the cotton samples.

Analyses of war experiences have shown that in World War I, most of the cases occurred during cold weather when woolen clothing was worn. In the recent Spanish Civil War where one of us (I. B.) had occasion to see and treat a large number of wounds, extremely few cases of gas gangrene were encountered. In China, in the Yangtze valley, where the soil is as richly manured as any land in the world, gas gangrene was uncommon. The same experiences were noted in Ethiopia. The common factor in the three countries mentioned above was the warm climate so that cotton clothing was worn.

There has been a report[5] of seven cases of gas gangrene on board a ship where soil could be excluded as a factor.

The predisposing factors are ragged penetrating wounds that severely damage muscle tissues, hematoma, or injuries to the vascular supply to the tissues. The buttocks, because they are easily contaminated by feces, and the lower extremities are the most common sites of gas gangrene. The upper parts of the body are uncommonly involved. Gunshot wounds of the retroperitoneal spaces that have traversed the rectum or colon are particularly prone to gas gangrene infection.

Symptomatology. In view of the fact that the gas bacillus causes hemolysis *in vivo*, the rapidly developing hemolytic anemia is explained and the peculiar discoloration of the skin, both locally and generally. The skin has a dirty-brown, yellow color, described as khaki colored, or it may even be blue tinged. The urine shows the characteristic color of methhemoglobinuria. The bacilli are sometimes recovered in the urine. Despite the fact that the organisms traverse the kidneys, there is slight or no damage to the kidneys except for an occasional pyelitis. Schottmueller and Bingold,[6] reporting on a large series of cases, found only two instances of diffuse nephritis resulting from gas bacillus infection.

There is only a moderate temperature rise and chills are rare. The pulse rate is increased out of proportion to the temperature. Exhaustion is marked.

Locally there is an increase of pain in the wound. In the early stages, slight edema around the wound is present, and paleness rather than redness is the characteristic appearance. Crepitus may be present but this may be a misleading sign. The discharge from the wound may be very profuse and of mousy or musty odor. It is watery or rust colored. With marked progress of the disease in the muscle, the discharge has the characteristic rotten-meat odor. *X*-ray examination of the involved area may aid in the diagnosis, showing the presence of air.

Bacteriologic examination of the wound shows the presence of organisms and these should be typed to determine the exact organism present in order to use the specific serum if possible.

Treatment. Treatment consists of surgery, administration of antigas-gangrene serum and chemotherapy. Wounds should be opened widely, incisions being made parallel to the long axis of the limb. The deep fascia is cut transversely. All involved muscle must be excised until normal bleeding muscle tissue is reached. The process must be truly a débridement if good results are to be expected. In the fulminating type of cases, only an amputation may save the patient.

Despite the controversy as to the efficaciousness of serum therapy, one of us (I. B.) has seen excellent results with serum therapy. Therapy should be adequate; 80 to 100 cc. of polyvalent serum in 1,000 cc. of saline should be given by the drip method. If the type of organism is known, monovalent serum is given. The

dose can be repeated. In an established case 400 cc. of the serum can be given within the first four or five days.

Of the sulfa drugs, sulfathiazole is the choice. Its action is best when placed into the wound at the time of operation. Experimentally, the use of concomitant oral administration does not seem to enhance the efficacy of the drug.

There have been some excellent reports of *x*-ray therapy for the treatment of gas gangrene. Since there are some differences of opinion, it would be better to await the results of more extensive use of this type of therapy.

ANTHRAX

This condition is caused by the Bacillus anthracis, and is transmitted from herbivorous animals to man, chiefly by goats, sheep, hogs and cattle. It, therefore, occurs chiefly in individuals whose occupations bring them in contact with these animals, such as stable workers, butchers, furriers and tanners. There have been numerous cases reported from infection through contaminated shaving brushes.

There are three portals of entry in the human being: The gastrointestinal tract is one portal of entry, but this is quite rare. The respiratory tract is another uncommon mode of entry. The broken skin is by far the most usual route of infection, and the exposed parts of the body such as the face, neck, hands and forearms are most frequently affected. The skin wound may be so slight as to escape detection.

The incubation period varies from twelve hours to five days, with an average of about two to three days. The first sign is that of a flea-bite like lesion which soon becomes papular and is brownish in color. This is surrounded by an area of erythema. Marginal vesicles appear about the lesion and the area is surrounded by a non-pitting edema. The brown center becomes black and hard, extending to the dried crusted vesicles and into the depth. The vesicles at first discharge a clear, gelatinous-like fluid which contains the anthrax bacilli. The discharge then becomes serosanguineous in appearance. The lesion is not painful, in contradistinction to carbuncles caused by the pyogenic organisms. Itching is a characteristic sign. The regional glands are involved in the majority of cases. They are painful and tender.

Systemic symptoms develop early, consisting of fever, fatigue and loss of appetite. In more severe cases, the systemic symptoms are much more in the foreground, consisting of vomiting, extreme exhaustion, high temperature, headache, pain in the joints, very rapid pulse, cyanosis, and often bloody diarrhea almost simulating that of cholera.

Treatment. The best results have been reported from the use of anti-anthrax serum and sulfa therapy. With proper treatment the mortality in cases of skin anthrax should be negligible.

Serum therapy has been well established. Large doses of anti-anthrax serum should be given and the dosage controlled by the recession of edema which is the best yardstick according to Gold.[7] An initial dose of 200 to 500 cc. of anti-anthrax serum should be given intravenously and repeated every twelve hours if the edema does not subside within eighteen to thirty-six hours. No serum should be injected about the local lesion. Persistently enlarged glands may be present even though the infection is under control. While pain and tenderness of the glands disappear early with successful treatment, persistent enlargement of the glands is no indication for further therapy.

Of the sulfa drugs, Gold[7] has found that sulfathiazole meets the requirements best. He gives an initial dose of 3 to 4 Gm. followed by 1 to 1.5 Gm. every four hours.

Comparing the results of serum with sulfonamides, he has found that chemotherapy has reduced hospitalization by slightly less than 50 per cent, and total disability by more than 50 per cent. If sulfa therapy does not control the edema within three days, shift treatment to large doses of serum.

SUMMARY AND CONCLUSIONS

Attention has been called to the important rôle played by minor skin infections in the causation of sepsis.

The systemic manifestations are those of septicemia, if caused by the staphylococcus, streptococcus or gas bacillus. The systemic symptoms are those of toxemia if the primary skin infection is due to tetanus or anthrax.

Staphylococcic sepsis, in which the portal of entry was through the skin, has been classified into five groups.

Streptococcic sepsis originating from the skin is rare. However, minor hair follicle infections, streptococcic furuncles or carbuncles, or streptococcic infections of a punctured wound of the skin acquired during the performance of a surgical operation or an autopsy, are the most common causes.

Since the introduction of sulfa drugs, the management of sepsis has been very encouraging, with a marked reduction in mortality.

It has been pointed out that the prophylactic treatment for tetanus by active immunization has already yielded excellent results, so that in the armed forces, where active immunization is universally practiced, the disease has almost disappeared.

The modern accepted treatment of gas gangrene and anthrax have been described in detail.

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REDUCTION AND AFTER-TREATMENT OF POSTERIOR DISLOCATION OF THE ELBOW

WITH SPECIAL ATTENTION TO THE BRACHIALIS MUSCLE AND MYOSITIS OSSIFICANS

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POSTERIOR dislocation of the elbow is a very common injury. Over a period of seven years at Massachusetts General Hospital, Wilson[10] reported elbow dislocation as the largest of any group of injuries of the elbow, exceeding even the common supracondylar fracture.

Posterior dislocation is usually caused by a fall upon the outstretched hand causing the elbow to become hyperextended. As hyperextension takes place, the olecranon process impinges against the olecranon fossa of the humerus and forces the lower end of the humerus past the coronoid process and through the anterior joint capsule into the brachialis muscle. Immediately following injury the patient usually flexes the elbow so that the surgeon first sees the elbow at an angle ranging from 90 to 135 degrees extension. There is usually a large amount of swelling about the elbow coming on within the first hour after injury.

The most common complications at the time of the injury include fractures of the medial epicondyle, the head of the radius, the olecranon and the coronoid process. The elbow is frequently also displaced laterally or, less frequently, medially. These complications are usually seen at the time of the injury and reduced at that time. It is a later complication,—myositis ossificans—that is most troublesome, often not anticipated and very frequently mistreated. Myositis ossificans is more common in the brachialis muscle than in any other muscle of the body.[7] It is also more common after posterior dislocation than any other elbow injury.[5,8] Because of the frequency of new bone deposit in the brachialis muscle, this muscle should receive the greatest amount of consideration in the reduction of posterior dislocation.

ANATOMY

The brachialis muscle is a large, fleshy muscle which originates from the anterior surface of the lower half of the humerus and inserts into the ulnar tuberosity and rough surface of the anterior coronoid process. (Fig. 1.) It is broad as it crosses the joint immediately anterior to the thin joint capsule, covering practically the whole joint and thus forming an important defense to the elbow joint.[4] Contrary to a common belief, the brachialis muscle does not form a tendon similar to the biceps but rather consists of both tendinous slips and muscle fiber bundles as it inserts. Many muscle bundles insert directly into the bone, especially the shorter fibers which originate lowest from the anterior and medial aspects of the humerus and insert on the proximal part of the rough surface of the anterior coronoid process. These muscle bundles lie directly anterior to the thin joint capsule. Thus, due to their anatomical position, they are especially vulnerable in posterior dislocation.

We have made cross-sections of the brachialis muscle in twenty different cadaver specimens directly opposite the humero-ulnar joint and attempted to determine as accurately as possible the percentage of muscle and tendon at this level where injury is likely to be most

severe. Planimeter measurements show that an average of 3.39 per cent of the muscle-tendon mass is tendon, the remaining 96.61 per cent being the soft vulnerable muscle belly. This offers striking contrast to the biceps which is practically 100 per cent tendon as it crosses the joint.

PATHOGENESIS OF MYOSITIS OSSIFICANS

Experimental production of posterior dislocation in the fresh cadaver specimen plainly shows the severe injury which affects the short fibers of the brachialis muscle. These muscle bundles are torn in their middle as well as many of the longer muscle bundles which are also torn and bruised.

It is not hard then to understand the pathogenesis of myositis ossificans, for, as Leriche and Policard[6] have so adequately pointed out, the ideal factors necessary for heterologous bone production are present: (1) the torn and bruised fibers lose their blood supply and degenerate; also, an intramuscular hematoma is formed; (2) calcium is available from periosteal stripping about the joint capsule attachments. In the presence of these two factors, preosseous substance is laid down.

The brachialis muscle is, therefore, a potential focus for new bone in every posterior dislocation and, although damage to muscle and periosteal stripping have already taken place at the time of injury, it becomes highly desirable to minimize additional trauma at the time of reduction.

REDUCTION

The usual method of reduction consists of extension and hyperextension of the elbow, followed by traction and flexion. It is thereby easy to secure a good bony reduction but only at the expense of the brachialis muscle. In reduction by this method, the ulna behaves as a second-class lever. As the elbow is extended to 180 degrees with the coronoid process in the olecranon fossa which acts as a fulcrum, force exerted at the end of the ulna is multiplied approximately ten times at the brachialis muscle. When the elbow is hyperextended with the olecranon against the posterior aspect of the humerus, there is roughly a five to one leverage system. A large amount of damaging stretch can thus be applied unthinkingly against the brachialis muscle.

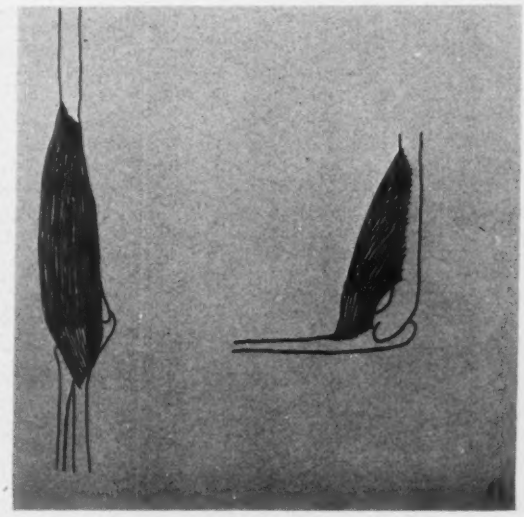

Fig. 1. The normal brachialis muscle is in a vulnerable position, especially the short fibers which originate lowest on the humerus and insert most proximally on the ulna.

Reduction by extension and hyperextension is said to be necessary in order to unlock the coronoid process from the olecranon fossa. Actually the coronoid process is only locked when the arm is in complete or nearly complete extension, and it can much better be unlocked in such a case by flexing the elbow to 90 degrees. Advocates of extension and hyperextension have asked the question, "How can re-extending and re-hyperextending the elbow do any more harm to the brachialis muscle than was done at the time of the injury, assuming that the injury was produced by hyperextension?" The answer to this question is obvious when it is realized that rarely is it possible to obtain a reduction within an hour after injury. By the time the patient reaches the surgeon and *x*-rays are completed, the elbow has changed. Marked swelling was present in all nineteen of our cases, even in those seen as early as one

hour following injury. Hemostasis in the muscle may be established by the time reduction is attempted. It is, therefore, apparent that repetition of extension and hyperextension is likely to start new hemorrhage. Also, traumatized muscle fibers may be in spasm by the time reduction is attempted so that the amount of extension and hyperextension which might have been harmless immediately after injury may be sufficient to damage further the spastic muscle fibers at the time reduction is attempted. Speed[9] pays tribute to this factor when he says that muscle spasm is the greatest obstacle to reduction of posterior dislocation.

Fig. 2. Mid-sagittal section through the elbow showing the distance in inches required for the muscle to stretch when reduced by flexion.

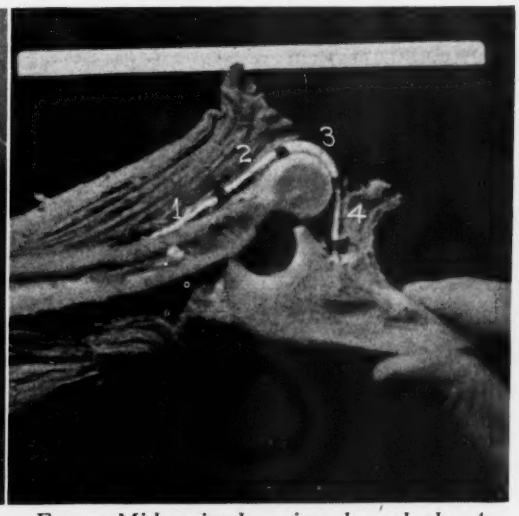

Fig. 3. Mid-sagittal section through the elbow showing the distance in inches required for the muscle to stretch when reduced by hyperextension.

We advocate reduction by flexion and traction of the elbow which in no way insults the brachialis muscle. (Fig. 2.) By this method the brachialis is relaxed as the arm is flexed to 90 degrees and needless stretch from extension and hyperextension is avoided. (Fig. 3.)

The exact method is in many ways similar to that of Bohler[1] and Crosby.[2] The patient presents himself to the surgeon holding his arm and his elbow at an angle of 90 to 135 degrees extension, usually about 125 degrees extension. The arm is splinted at 90 degrees flexion with the forearm in mid-pronation and analgesia given while *x*-rays are taken. If the diagnosis of posterior dislocation is confirmed by *x*-ray, anesthesia is administered. General anesthesia is preferable although local or brachial plexus block may be used. The splint is removed and any lateral or medial dislocation is corrected. Strong traction is then applied to the forearm, keeping the elbow at 90 degrees flexion. Countertraction is furnished by an assistant who grasps the upper half of the humerus or the patient may be strapped to the table using a heavy felt axillary pad. The forearm is kept in mid-pronation during traction to relax the pronator teres which is usually injured in posterior dislocation.[1] The operator uses both hands in applying traction, one hand being at the wrist and the other hand near the elbow. (Fig. 4.) Following traction the elbow is flexed to roughly 70 degrees and maintained in flexion with one hand at the wrist while with the other hand strong pressure is exerted on the anterior aspect of the lower humerus which accomplishes reduction. (Fig. 5.) It is important that the elbow be flexed beyond 90 degrees before pressure is exerted on the humerus, for in such position the coronoid process will not be fractured. (Fig. 2.)

AFTER-TREATMENT

A review of the literature of the past fifteen years reveals a tendency for the most part to immobilize the injury after reduction in a position of hyperflexion for two to three weeks. However, two papers[3,11] have appeared during this time advocating motion as early as two days following injury. We do not believe in early motion.

Whether new bone is laid down around the capsule or in the brachialis muscle, resorption of the excess new bone is greater if the extremity is immobilized.[6] An understanding of the pathogenesis of excess new

Fig. 4. The elbow is flexed to 90 degrees to relax the brachialis muscle and traction is applied to the forearm in mid-pronation to relax the pronator teres.

Fig. 5. The elbow is flexed beyond 90 degrees while pressure is exerted on the lower humerus to avoid fracture of the coronoid process.

Fig. 6. With the elbow flexed beyond 90 degrees the arm is placed in a posterior plaster of Paris splint until swelling subsides.

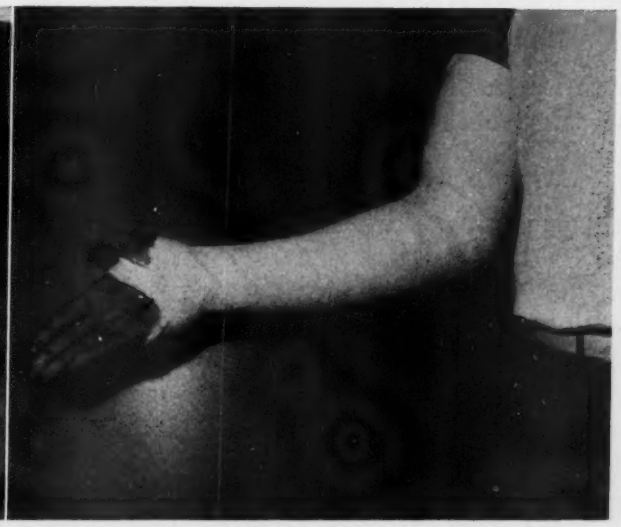

Fig. 7. The completed unpadded cast is trimmed around the thumb and back to the proximal palmar crease to enable the patient to use his hand.

bone helps to clarify this statement. A mass of new bone in muscle is an obstruction to normal muscle function. Normal muscle fibers adjacent to the calciferous deposit are repeatedly traumatized and offer new foci for more new bone formation if motion is allowed. The process is, therefore, a vicious cycle which can only be interrupted by immobilization. Equally important is the maintenance of a good blood flow through the injured extremity since good circulation will also hasten the resorption of excess new bone.[6]

With these two facts in mind, we have adopted the following treatment. After reduction of the posterior dislocation, a posterior plaster of Paris splint is applied with the elbow at about 80 degrees flexion, provided the radial pulse is satisfactory at this angle. (Fig. 6.) The forearm is placed in mid-pronation as previously mentioned, to relax the pronator teres. The injured elbow is elevated 45 degrees above the body level for at least forty-eight hours and the radial pulse checked frequently. At the end of a week if the swelling around the elbow has reached a minimum, a light unpadded plaster of Paris cast is applied with the elbow at the position of function (90 to 110 degrees). The cast should extend from the axilla to the metacarpophalangeal joints on the dorsum of the hand and must be trimmed around the thumb and in the palm back to the proximal palmar crease. (Fig. 7.) The patient can thus carry out his work in most instances. A lightweight unpadded cast is essential as the patient will not use his arm with a heavy cast. The patient is advised to use his hand with the cast on, to carry out his occupation if possible and not to carry the arm in a sling. He is told to place the arm over the head and behind the back twenty times daily which prevents atrophy of shoulder muscles. Weekly visits are essential to make sure that these instructions are carried out. The cast is removed in three to four weeks and *x*-rays taken for excess new bone in the brachialis muscle or around the capsule. If this is found, immobilization in plaster must be continued until the calciferous mass disappears.

We have, therefore, attempted to meet the requirements for the resorption of excess new bone by providing a light unpadded cast which can be well molded to the arm, gripping the skin creases as well as the hairs of the arm. A good flow of blood is continually pumped through the injured extremity as the patient uses the hand. The functional advantages are many. When the cast is removed, there is little or no muscle atrophy; the hand grip is strong, the muscle tone and skin texture good. The elbow usually has 60 to 90 degrees range of motion upon removing the cast. Active motion is easily begun and there is less need for physiotherapy. All of our patients have carried out their occupations while wearing their casts and the economic advantages are thus evident.

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ACUTE PERFORATED DUODENAL ULCER

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THE ever present threat of perforation is a deadly menace to the patient who has a duodenal ulcer. The incidence of perforation is not known but it seems to have geographic variations. It is more frequent in England than in this country. A chronic duodenal ulcer may be present for eight or ten years and suddenly perforate or perforation may be the first and only symptom. As yet, there is no explanation for this unusual behavior. There is very little assurance in the prevention of perforation by medical treatment. However, perforation of a duodenal ulcer is extremely rare after gastroenterostomy because the original ulcer almost invariably heals.

Acute perforation of a duodenal ulcer necessitates an immediate operation. There are no recognized reasons for delay. The obvious surgical procedure is closure of the perforation. Whether this should be supplemented by gastroenterostomy is a question on which there are different opinions. Statistics do not afford the answer. The figures for surgical mortality vary and depend on many factors; the one most commonly stressed is the interval of time between perforation and operation. Cultures of the peritoneal fluid in the early hours after perforation usually are sterile—probably the result of the action of hydrochloric acid in the stomach. Later the cultures often show pathogenic bacteria and there is evidence of peritonitis. The nature of the contents of the stomach may or may not have a bearing on the virulence of the peritoneal process. Patients with duodenal ulcer usually have an abundance of free hydrochloric acid in the stomach but there is no available information as to the length of time required for ingested fluid to be rendered free of bacteria by the action of the acid; or, for that matter, if it ever becomes sterile. It may be assumed that after a meal has been completely acted upon by the stomach the resultant mixture is sterile, but surely it is not uncommon for food stuff to leave the stomach and enter the duodenum before the above process is complete.

Casting theory and speculation aside, it stands as a fact, as in many other surgical conditions, the earlier the operation the lower the mortality. Without surgical help, these patients usually die of peritonitis, which is also the most common cause of death after operation. The operative procedure may be accused of contributory negligence if a duodenal fistula or a subphrenic abscess occurs. No patient with acute perforation of a duodenal ulcer, regardless of delay or degree of peritonitis, should be denied the chance which only operation can offer. Surgery is subject to a multitude of human diversities. No two "cases" are alike. The mortality of an operation in a thousand reported cases may be 5 per cent but for the patient who dies it is 100 per cent. A competent surgeon may encounter a series of fatalities because of uncontrollable circumstances and a tyro may have a number, large or small, of the same type of case without a death. After all, is it not true that the surgeon when confronted by a problem in the individual patient is by duty bound to do the right thing at the right time and let statistics fall where they may? To do this requires, of course, training and experience, and that which is more important, observant and thoughtful consideration of pathologic states and physiologic changes.

The mortality of operation in acute perforation, before twelve hours, should be under 10 per cent; after that interval it rapidly increases. Peritonitis is the most frequent cause of death even in those patients who succumb after an early operation. The peritoneal fluid is not always sterile in the first four or five hours after perforation and it is not always true that early operation means recovery. Death may be the result of subphrenic abscess and pulmonary complications.

Is it not an obvious fact that all duodenal ulcers are potential cases of perforation? It is impossible to separate into clinical groups ulcers which will bleed, ulcers which will perforate, ulcers which will cicatrize and stenose, and those which will run a smooth uneventful prolonged course. There is no basis in fact for the assertion that bleeding ulcers do not perforate. The natural history of duodenal ulcer is characterized by alternate healing and reactivation. Usually these phases correspond with clinical symptoms. Ulcers which have no tendency to heal do not show scar tissue. It is rare for the surgeon to expose a chronic duodenal ulcer without evidence of scar tissue. An exception is duodenitis in which a definite ulcer is absent. In the picture of an acute perforation of the duodenum there is usually very little if any evidence of scar tissue to indicate the presence of chronic ulceration. I have no positive conviction that only acute ulcers perforate but in my experience the findings at operation and the frequent absence of ulcer symptoms prior to perforation strongly suggest it. In not a few cases of duodenal ulcer the first symptom is either from perforation or hemorrhage. It is significant of an acute process that some patients experience a short preperforative period of one to three weeks in which the symptoms of ulcer are intense and constant and the usual methods of relief as by antacid powders and diet are of no avail. In these patients perforation seems to be inevitable, but usually one is unable to foretell this catastrophe. Medical treatment does not prevent, and I seriously doubt that it even reduces the incidence of perforation. There are authentic records of patients who have suffered a perforation while confined to bed under hospital treatment. One of my patients had an acute perforation within a few minutes after a barium meal for an *x*-ray examination of his stomach.

The effect of injury or strain of the abdominal wall as a cause of rupture of an ulcer has received occasional comment. One of my patients, a structural iron worker, had an acute perforation of a duodenal ulcer while he was holding a rivetting hammer against his abdomen. Of course, this may have been a coincidence and not cause and effect. However, there are many instances on record in which perforation followed so close upon muscular effort associated with lifting, cranking a motor and in some cases direct blows to the abdomen, that an etiologic relationship may not be unlikely. I suspect that the general concept of the process of perforation of a duodenal ulcer is a progressive erosion through the coats of the duodenal wall until the protective covering on the peritoneal side becomes so thin that, like the blowout of a worn automobile tire, sudden explosive rupture occurs. Anyone who has given some thought to the appearance of a duodenal perforation at the operating table knows the utter fallacy of the above theory. The picture of the perforation is nearly always the same. It is not a tear nor is it a blowout. There is nothing about it to suggest physical force from without or from within. It is always a clean-cut rounded hole. A quiescent or healed ulcer does not perforate nor rupture. The evidence indicates that acute perforation occurs only in an acute ulcer or in a chronic one which has undergone acute exacerbation. The actual perforation is the result of an active ulcerative process which usually is rather rapid from start to finish because one rarely sees protective adhesions.

The recognition of an acute perforation of a duodenal ulcer is so easy that it is difficult to explain the long delay which often occurs before operation. Unlike the symptoms of acute appendicitis those of perforated ulcer are dramatic and faithfully characteristic. I saw a patient within a few minutes after his ulcer perforated. He had severe upper abdominal pain and localized tenderness but rigidity of the muscles had not yet appeared. There was not the slightest indication of shock and the pulse and temperature were normal. At operation, less than two hours later, there was an opening about 1 cm. in diameter in the duodenum from which bile-stained fluid was pouring into the free peritoneal cavity. Within a short time after perforation the pain becomes agonizingly severe; it is constant and without remission. At the same time there develops a rigidity of the abdominal muscles, more pronounced in the upper part, which is board-like and serves to splint and to immobilize the entire abdomen. In no other condition is there rigidity such as this; it is the most characteristic physical finding. The patient remains flat on his back and is afraid to change from this position. Vomiting is not common. Grunting respiration is a characteristic symptom. A flat *x*-ray plate of the abdomen for detection of an air bubble under the diaphragm is said to be of diagnostic importance. It has been my experience in the surgical diagnosis of acute abdominal conditions that the more laboratory aids are brought into use the more likely it is for confusion to replace clear thinking and the patient becomes the loser as the result of indecision and delay. There is one forgivable error in the diagnosis of perforated ulcer—acute perforative appendicitis. After the lapse of a few hours the fluid from the perforation follows a natural drainage path as pointed out by Moynihan years ago, down the outer side of the right colon, over the region of the appendix and into the pelvis. In such cases, usually late, there is tenderness and rigidity over the area of the appendix and often a tender pelvic mass by rectal palpation. Fortunately, these findings usually call for immediate operation. Once the abdomen is opened, the character of the peritoneal fluid noted and the appendix examined, the observant surgeon will readily recognize the situation and act accordingly; thereby no harm has come to the patient.

Occasionally, one will see a patient ten or twelve hours after the onset of sudden and severe upper abdominal pain. At present he is sitting in a chair and he is able to walk about the room. The pulse and temperature are normal. The only complaint is epigastric soreness. Palpation of the abdomen detects board-like rigidity which is confined to the right upper quadrant and firm pressure elicits deep tenderness. It is difficult to convince him that he should submit to operation. Exploration reveals a perforated duodenal ulcer which has become loosely sealed to the undersurface of the liver. The attachment is so insecure that it lets go with the slightest manipulation and it is a great satisfaction to the surgeon that he is at once prepared to prevent an inevitable disaster. The above circumstances occurred in a patient upon whom I operated eighteen hours after perforation. He recovered promptly after closure of the perforation and a posterior gastroenterostomy, and has remained entirely well to date now eight years after operation.

The method of anesthesia is of great importance but it has been consistently ignored or unrecognized in discussions of the subject. The anesthetist often has difficulty in maintaining a smooth anesthesia with gas or ether, and in such instances postoperative pulmonary complications seem to be more frequent. Of greater importance is the almost constant difficulty in obtaining relaxation of the abdominal muscles so that it becomes necessary for the surgeon to fight his way through guarded muscles and the cantankerous annoyance of protruding abdominal contents. Under these conditions the handling of a grave surgical lesion

which requires impeccable technic may be an extremely difficult task. Is it not possible that the above situation, not always conducive to tranquillity of mind and procedure, has had something to do with the oft repeated admonition that these patients are desperately ill, that only emergency treatment, namely, closure of the perforation is justified and that gastroenterostomy subjects the patient to undue prolongation of the operation? Complete relaxation can be obtained with spinal anesthesia which I have used in these cases for sixteen years. Under its influence the field of operation is quiet, there is less traumatism to peritoneal surfaces; accurate technic is favored; the surgical procedure can be carried out in an unhurried manner and an additional fifteen minutes for a gastroenterostomy does not increase the operative risk. A patient with a perforated duodenal ulcer, other things being equal, stands as good a chance for recovery after forty-five minutes on the operating table as another similar patient who has been there only thirty minutes. Operations many times are prolonged by clumsy operating, unnecessary maneuvers, needless handling of tissues and failure to complete one technical detail before moving on to the next. In my experience spinal anesthesia has been more satisfactory than any other method and equally safe.

The preoperative diagnosis usually is correct and the incision properly placed. When through error of diagnosis (appendicitis) the incision has been made in the right lower quadrant the true state of affairs is soon revealed. Remove the appendix and close the incision; then make a separate incision in the right upper quadrant. Occasionally, it may be wise to use the lower incision for the insertion of a drain. When the peritoneum is opened, the diagnosis is confirmed by the appearance of bile-stained fluid which contains flakes of plastic exudate and often small bits of food débris. In many instances gas bubbles are seen. The fluid should be mopped up with large moist gauze sponges or removed by suction. The stomach is then pulled downward and to the patient's right, handed to an assistant who holds it with a dry sponge and turns it toward the patient's left. By this maneuver the site of perforation in the duodenum is freely exposed and the parts are under perfect control for closure of the perforation. Moist gauze pads should be judiciously placed to prevent further soiling of the peritoneum. During these manipulations one must be exceedingly careful to prevent the free fluid from gaining access to the space between the uppersurface of the liver and the diaphragm. This region is difficult to cleanse properly and methods for draining it are inadequate. The danger, of course, is the development of a subphrenic abscess. It is remarkable how quickly plastic exudate forms after a perforation of a duodenal ulcer; it is not uncommon to find the perforated part of the duodenum loosely adherent to the undersurface of the right lobe of the liver. Leakage from the opening rarely is completely checked but usually materially lessened, so that the spreading of peritonitis is temporarily halted and localized. I have never seen the great omentum taking part in sealing over the perforation. In many instances, on the other hand, it is not uncommon to expose a perforation which is as free as a leak in a rainspout and pouring forth fluid containing particles of undigested food which may be infested with bacteria. It is obvious that the interval of time between perforation and operation in the first instance is comparatively unimportant while in the latter it becomes the chief determining factor in prognosis. Such factors, and they are rarely mentioned, have a profound effect upon statistics and on the patient.

Free perforation of a duodenal ulcer presents a characteristic picture at the operating table. Invariably there is a round pyunched out opening 5 to 8 mm. in diameter and about 2 to 4 cm. distal from the pylorus on the antero-superior surface of the duodenum. For a distance of 2 or

3 cm. around the opening the duodenum shows the grey pallor of inflammatory edema; its wall is much thicker than normal and has lost its pliable, supple quality. Because of these local tissue changes sutures easily tear out. Infolding of the perforation is almost impossible and secure accurate closure as we would deem a necessity for an opening elsewhere in the bowel cannot be accomplished.

With the duodenum adequately exposed and the perforation visualized, it is closed by three or four through-and-through cotton sutures which are placed longitudinally or transversely depending upon the ease of closure. Several pieces of nearby mesenteric fat or omental tabs are included by a third knot in the above sutures, thereby making the closure more secure against leakage. No effort is made to infold the perforated area and only one layer of sutures is used. Only enough is done to close the opening because too much suturing is likely to favor leakage and to weaken the closure. After the perforation is exposed and before the closure is made, it is my practice to clean out by the use of large moist sponges and suction all accessible free fluid. Large moist gauze pads are then placed around the area of perforation, a pad in the subhepatic space, another pad between the undersurface of the liver and the duodenum which serves to prevent contaminated fluid gaining access to the space between the liver and diaphragm, and another moist sponge is placed just above the duodenum. I believe that the proper disposition of gauze in operations for perforated duodenal ulcer is second in importance only to a similar precautionary measure in cases of acute perforated appendicitis. Too often, fatal peritonitis is man-made. A posterior short-loop gastroenterostomy is now made. My reasons for doing a posterior gastroenterostomy are based on the following personal convictions. A duodenal ulcer is a local manifestation of disturbed physiology of the stomach; acute perforation of the ulcer is a complication and not a cure of the disease. Closure of the perforation alone may save the patient's life but he is left in a state of potential danger of another perforation. The high rate of recurrence of duodenal ulcer after local excision certainly does not support the theory that perforation cures the ulcer. On the other hand, the rate of recurrence of ulcer in the duodenum following gastroenterostomy is very low. At least two-thirds of the patients who have had a simple closure of an acute perforation experience symptoms indicative of recurrence of the ulcer. Does the addition of gastroenterostomy increase the operative risk? I believe that the operation in competent hands actually is a safety measure so far as the immediate condition is concerned. Certainly the additional fifteen minutes does not increase the risk, so that, the only factor of danger would be some technical error in the operation itself. The performance of gastroenterostomy affords definite protection to the recently sutured opening in the duodenum. Under the best conditions this closure is not secure. The resumption of traffic over the weakened and partially devitalized area immediately after suture and the added physiologic distention resulting from forceful ejection of contents from the stomach make the margin of safety very narrow from the standpoint of leakage. In cases of death from peritonitis or from subdiaphragmatic abscess after simple closure of a perforated ulcer, is it not probable that leakage at the suture line of closure has been at fault? The necessity of a gastroenterostomy should not depend upon any apparent narrowing of the lumen of the duodenum incident to closure of the perforation. After the inflammatory edema has subsided and healing has occurred it will be found, as it usually is at subsequent operations, that the duodenal lumen is entirely adequate. I believe that the making of a gastroenterostomy will prevent or lessen the dangers described in the preceding remarks. I have known of several instances in which duodenal fistulas followed simple closure of a perfora-

tion although I am not prepared to say that the complication would have been prevented by a gastroenterostomy. It is reasonably certain that for a varying length of time after closure of an acute perforation there is more or less physiologic or anatomic obstruction to the emptying of the stomach. I have performed gastroenterostomy in five patients for recurrence of duodenal ulcer several years after simple closure of an acute perforation. In these cases the duodenum and pylorus were inseparably adherent to the undersurface of the right lobe of the liver, thereby constituting a mechanical fault as far as the function of the pylorus was concerned. Without attempting to separate the adhesions, a careful inspection revealed inflammatory changes with evidence of the presence of active duodenal ulceration. Is it not probable that by sealing the duodenum to the undersurface of the liver nature had done more than the surgeon to close the perforation and to prevent a spreading peritonitis? At any rate the mechanical derangement of the pyloric region of the stomach incident to this condition even in the absence of active duodenal ulceration would be a sufficient reason for the making of a gastroenterostomy. Then, too, it is possible for this mechanical derangement of the pylorus actually to favor the recurrence of duodenal ulceration. The patient with a duodenal ulcer has a constitutional disorder of the function of the stomach which will continue to form ulceration in the duodenum regardless of whether the ulcer is perforated or surgically excised. In a small percentage of cases, this constitutional defect favors the formation of an ulcer in the stoma of the anastomosis when the gastroenterostomy has failed in its primary purpose, namely, the control of excessive gastric acidity.

It is a well known fact that slow, pinpoint perforation of a duodenal ulcer rarely gives rise to spreading peritonitis. The slowness of the leakage permits ample time for the formation of protective adhesions to nearby structures. Usually, these patients have had a duodenal ulcer for years and then rather suddenly the mild intermittent symptoms of ulcer are replaced by constant localized epigastric pain. It may be severe but it is never so acute as when free perforation has occurred. There is no board-like rigidity of the abdomen, only localized tenderness. Gastric retention and vomiting may be present. Does not this situation call for urgent surgical relief? Would it be sound surgical judgment to separate the adhesions which seal the perforation to the undersurface of the liver and then to close the perforation and let it go at that? I believe that nearly every surgeon would leave the local condition strictly alone and consider the situation an ideal indication for the making of a gastroenterostomy. If gastroenterostomy is indicated for this kind of perforation, why should it not be equally applicable in the case of acute free perforation? It seems to me that the only logical reason for the omission of gastroenterostomy in the case of acute perforation is the alleged risk incident to the prolongation of the operation, especially in the presence of advanced peritonitis. The omission of gastroenterostomy may be justifiable on the grounds of expediency, but imperfect postoperative results are likely to follow in 40 to 60 per cent of the patients. In this group there will be instances of second perforation and the majority will require gastroenterostomy because of the persistence of the symptoms of duodenal ulceration. These events will carry their own risks and mortality figures. After all is said and done, gastroenterostomy in spite of its faults, failures and misuses remains the most satisfactory method of treatment in properly selected cases of duodenal ulcer. This is true because it insures nature's own control of the vicarious acidity of hypersecretion which amounts to a constitutional fault in the ulcer bearing patient. This fault of gastric chemistry is part of the patient's makeup and as such is intractable and persistent. To ignore this tenet in the treatment of a perforated

duodenal ulcer is to invite recurrence. The present state of development of surgical technic demands in the case of the patient with perforation not only that his life be saved by closure of the perforation but that his future be relieved of the anxiety and ill health incident to the lesion which originally laid him low.

from the surrounding territory. Many patients have died of peritonitis in spite of drainage but I do not know of any instance in which death from peritonitis has been attributed to the failure to insert a drain. There are fewer indications for the suprapubic drain than there are for a drain through the operative incision. A word of

TABLE I

		Age	Sex	Duration of Perforation	Operative Procedure				Anesthesia	Deaths
					Closure of Perforation	Gastroenterostomy	Appendectomy	Drainage		
1	D. W.	33	M		*	*	*	o	Spinal	
2	D. B.	57	M		*	*	*	o	Spinal	
3	J. K.	22	M	5 hrs.	*	*	*	o	Spinal	
4	E. L.	59	M	14 hrs.	*	*	o	o	Spinal	
5	J. S.	32	M	4 hrs.	*	*	o	o	Spinal	
6	S. F.	42	M	6 hrs.	*	*	*	o	Spinal	
7	C. B.	30	M	6 hrs.	*	*	o	o	Spinal	
8	R. B.	33	M	8 hrs.	*	*	*	o	Spinal	
9	J. F.	34	M	5 hrs.	*	*	*	o	Spinal	
10	G. B.	49	M	4 hrs.	*	*	o	*	Spinal	
11	M. A.	30	M	2½ hrs.	*	*	*	o	Spinal	
12	T. B.	38	M	18 hrs.	*	*	o	o	Spinal	
13	J. F.	46	M	5 hrs.	*	*	o	o	Spinal	*
14	J. W.	18	F	12 hrs.	*	*	*	o	Ether	
15	J. X. P.	47	M	5 hrs.	*	*	o	*	Spinal	
16	E. S.	19	M	10 hrs.	*	*	*	o	Spinal	
17	J. Mc. A.	43	M	7 hrs.	*	*	*	o	Spinal	
18	R. M.	33	M	14 hrs.	*	*	*	o	Spinal	
19	E. P.	24	M	8 hrs.	*	*	*	o	Spinal	

* = yes.
o = no.

The matter of drainage after operation for acute perforation cannot be settled by statistical study, nor can rules of procedure be laid down. Usually the insertion of a drain does no harm, but that does not constitute a reason for using it. If one is uncertain about the security of the closure of the perforation and has not done a gastroenterostomy, the use of a drain may be a safety procedure. There is always peritoneal contamination in these cases but if one must use a drain on this basis, where should it be placed? Bear in mind that a drain only serves to form a sealed-off outlet to which there are no tributaries

warning should be said regarding the placement of the drain when it is used. To place the end of the drain, whether it be of the cigarette type or a plain rubber tube, down to and possibly in contact with the site of the closed perforation in the duodenum is a dangerous procedure because it favors or may actually incite leakage from the suture line. I suspect in many cases in which an apparent laudable purpose has been served by the drain, so that the surgeon could point with pride to his justifiable practice, that if the truth were known there would have been no drainage or leakage if the drain had been omitted.

SUMMARY

This report is based on a personal experience with nineteen patients who were operated upon for acute perforation of a duodenal ulcer. Closure of the perforation and posterior gastroenterostomy was done in all and in twelve the appendix was removed. Drainage was used in only two cases. There was one death. This patient who was admitted in a state of shock five hours after perforation and operated upon at once died twenty-four hours later. The youngest, and only female in the group, was a girl of eighteen years. This is not a selected group but one which contains all cases of acute perforated duodenal ulcer which have come under my care in the past fifteen years.

Many perforated duodenal ulcers become sealed temporarily to the undersurface of the liver, thereby preventing for a time widespread contamination of the peritoneum.

Acute perforation is the result of an acute ulcerative process in either a recent or an old ulcer. The perforation is caused by erosion (acid) and not by rupture from increased pressure.

The operation of choice should be closure of the perforation and posterior gastroenterostomy. The closure is made by three or four through-and-through cotton sutures re-enforced by several nearby fat tabs. Do not infold; do not use a purse-string suture.

Except in the presence of advanced peritonitis, gastroenterostomy is advisable because (1) perforation does not cure the ulcer. Simple closure is followed by recurrence of ulcer in 40 to 60 per cent of the patients and in others the stomach may have motor difficulty from fixation of the pylorus to the undersurface of the liver. Gastroenterostomy is the best safeguard against recurrence and motor dysfunction of the stomach. (2) It protects the sutured area of perforation against tension and leakage. (3) It does not increase the operative risk.

When the appendix is readily accessible, it should be removed. Spinal anesthesia is recommended except in the presence of shock. Drainage is rarely indicated.

REPAIR OF URINARY BLADDER HERNIATION

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ONE of the most difficult varieties of hernia to repair is that in which the minor or major part of the content is a protruded bladder wall. This may be either of the primary or the secondary form.

The chief objective in the surgical correction hereinafter described is, manifestly, a permanent cure.

Hernia of the bladder was first referred to by Abulcasis in the twelfth century, and by Guy de Chauliac (1363). The latter surgeon described the technic of the passage of a catheter as an aid in diagnosis. The first recorded case was that of G. D. Sala, of Padua (1620). Plater (1550) also described a bladder hernia.

INCIDENCE

Urinary bladder hernia is either a complication or an integral part of an inguinal hernia. Various statistical studies reveal an incidence of 1 to 3 per cent, but in my experience it is at least 10 per cent in patients over fifty years of age with inguinal hernias of even moderate size.

In recurrences following inguinal herniopplasties some surgeons give a percentage as high as 25.5.

In 1935, I summarized my views regarding this subject, to the effect that the so-called recurrences after operations for inguinal hernia are not, as a rule, true recurrences which reveal a definite reformed sac, but are more frequently either a return of the condition imperfectly repaired or a postoperative incisional inguinal hernia. In either instance the protruding structure is frequently the bladder. Incisional hernias occur in an older age group with poor musculature and obesity; this must be borne in mind at the first surgical procedure as re-operation usually produces another so-called "recurrence."

The fundamental cause, in the past, was timidity on the part of the surgeon to open the bladder protrusion and also failure to analyze the anatomic basis for the condition, which, in reality, was present prior to the first attempt at surgical repair.

VARIETIES

Three main anatomic varieties of bladder herniation have been described; (1) Intraperitoneal—the bladder diverticulum is a component of the hernial sac; (2) paraperitoneal—the hernial protrusion is covered peritoneally only on one side (the most common variety); (3) extraperitoneal—when a sac is present, the bladder is distinct from, and medial to it.

This classification, though accurate, is inadequate and is not descriptive enough. Therefore, I offer the following five types, based primarily on a surgical-anatomical foundation (Fig. 1):

1. As part of a direct inguinal hernia through Hesselbach's triangle, without evidence of an indirect inguinal sac. The size varies from a protrusion about an inch in diameter to one of several inches; the base is sessile and covered by an attenuated transversalis fascia; the inferior epigastric blood vessels may be partly displaced laterally. (Figs. 1D, 2 and 3.)

2. As a complication of an indirect inguinal hernia in which there is no direct connection between the bladder protrusion and the indirect inguinal component. (Figs. 1E and 4.)

3. As the medial part of a large saddlebag or pantaloon hernia in which the lateral bladder wall protrusion is part of

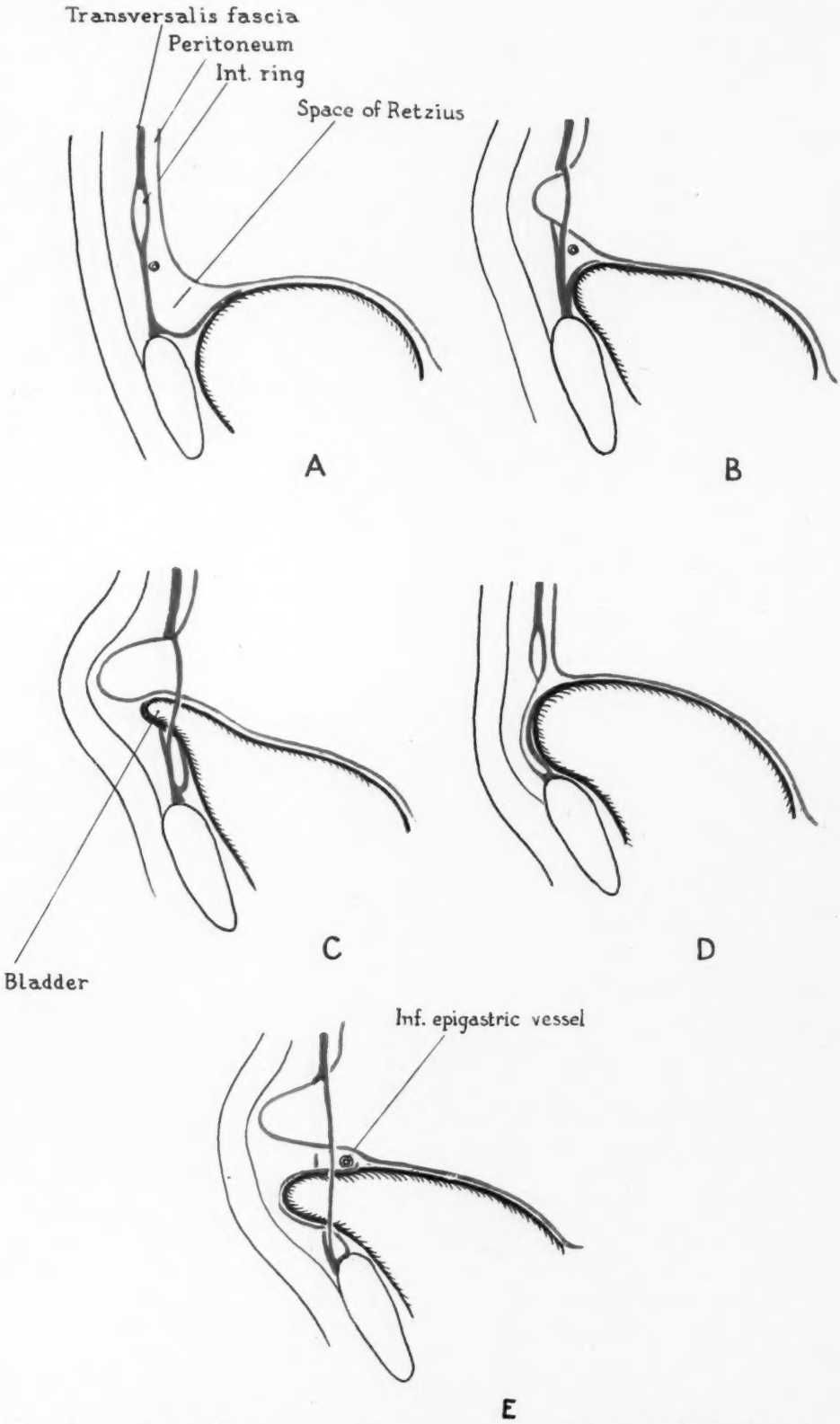

FIG. 1. A, normal relationship of peritoneum, transversalis fascia and bladder. B, small indirect inguinal hernia showing dilated deep ring and effect on bladder wall. C, later stage showing bladder hernia complicating indirect inguinal hernia. D, bladder hernia; no evidence of indirect inguinal hernia. E, saddle-bag or pantaloon hernia; the bladder forms the medial protrusion.

the medial wall of an indirect hernial sac. (In this variety, as in the two preceding, the transversalis fascia is attenuated to an extreme degree and the only dividing line between the bladder protrusion and the indirect hernial sac is a cleft formed by the inferior epigastric vessels.) Here, the bladder hernia is enlarged owing to peritoneal traction or tension as the indirect sac grows. The urinary bladder wall enters the hernial sac in one of two ways: A part of its wall fuses with the medial wall of the hernial sac so that the bladder proper, in effect, forms one of the sac layers. The condition is, in reality, a sliding hernia of the blader. This is especially true of massive hernias. On rare occasions the intraperitoneal part of the bladder lies directly within the complete hernial sac. (Figs. 1C, and 5.)

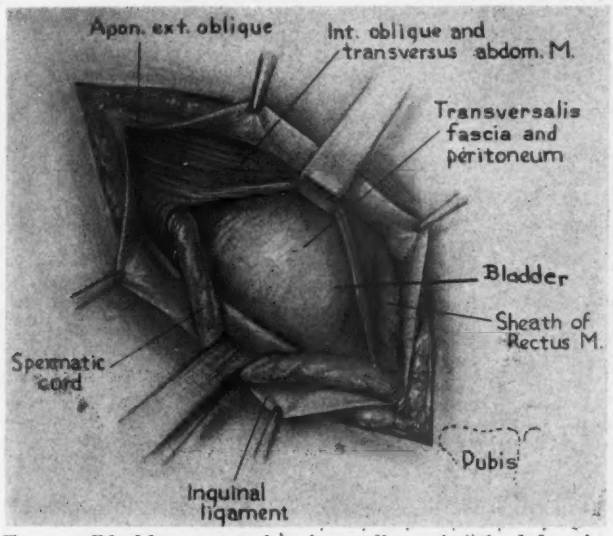

Fig. 2. Bladder protrusion in a direct inguinal hernia.

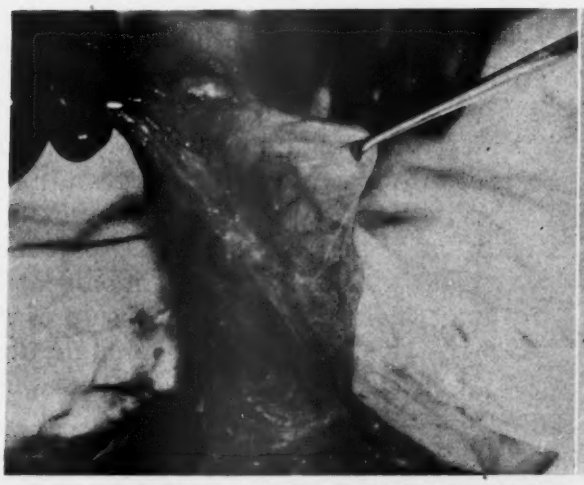

Fig. 3. Large direct right inguinal hernia; no evidence of an indirect hernia. The bladder constituted the entire hernia.

Fig. 4. Saddle-bag or pantaloon hernia—type 3.

4. Recurrent inguinal hernia (so-called) is frequently an incompletely repaired saddlebag or direct inguinal hernia. (Fig. 6.)

5. Femoral bladder hernia. (Fig. 7.)

The following varieties of bladder hernias have been reported in the literature on extremely rare occasions: (1) Obturator (extraperitoneal); (2) suprapubic—through

the linea alba; (3) ischiorectal, and (4) Gironcoli's—a protrusion directly through the rectus abdominis muscle. The ring is the rectus sheath and the muscle.

Fig. 5. Saddle-bag hernia, showing two sacs; the smaller one is the bladder.

ETIOLOGY

Bladder herniation is more frequent in men and in both sexes shows a distinct predilection for the right groin. The condition may be associated with a hernia of other organs on the same or opposite side of the body.

Fig. 6. Bilateral recurrent inguinal hernia. At operation right side was repaired. There was a "recurrent" bladder hernia.

The age factor as well as prostatic enlargement are of great importance in the formation of the hernial disability. Arduous work or a chronic cough over a long period of time, aggravated by severe bodily strains, are additive and significant factors. Asthenia, following long illness, is another predisposing cause.

Aside from the ordinary causes, there are intrinsic changes in the bladder musculature, with increased urinary pressure due to urethral or prostatic obstruction. A condition of permanent vesical distention may exist with a consequent modification of the normal anatomic relationship of the bladder. The organ is thus brought into direct contact with the inguinal fossa. The vesical wall then becomes attenuated.

There is, as a rule, a history of a long-standing, large, irreducible hernia which perceptibly diminishes in size after urination or catherization.

The herniation may occur through the inguinal or femoral rings and sometimes, as aforementioned, through the obturator foramen or other apertures.

SYMPTOMS

Urinary symptoms, oddly enough, are not always characteristic features of bladder herniation. If urinary symptoms are

present (dysuria, frequent or infrequent urination, and unusual tenderness over the protrusion), they are usually due to complications. The patient urinates at intervals, or only when he assumes a certain position or presses on the tumor mass. The last named indication is highly suggestive of the condition. The gastrointestinal symptoms are mainly of reflex nature. The severity of the symptoms manifestly depends on whether or not there is an associated strangulation, obstruction or other complications.

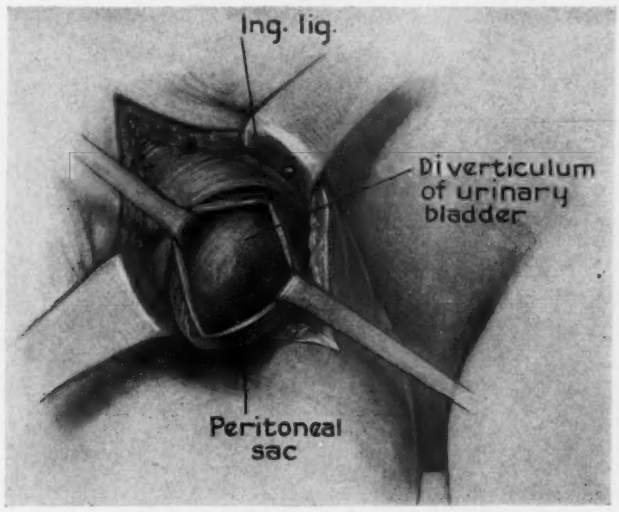

Fig. 7. Bladder protrusion in femoral canal.

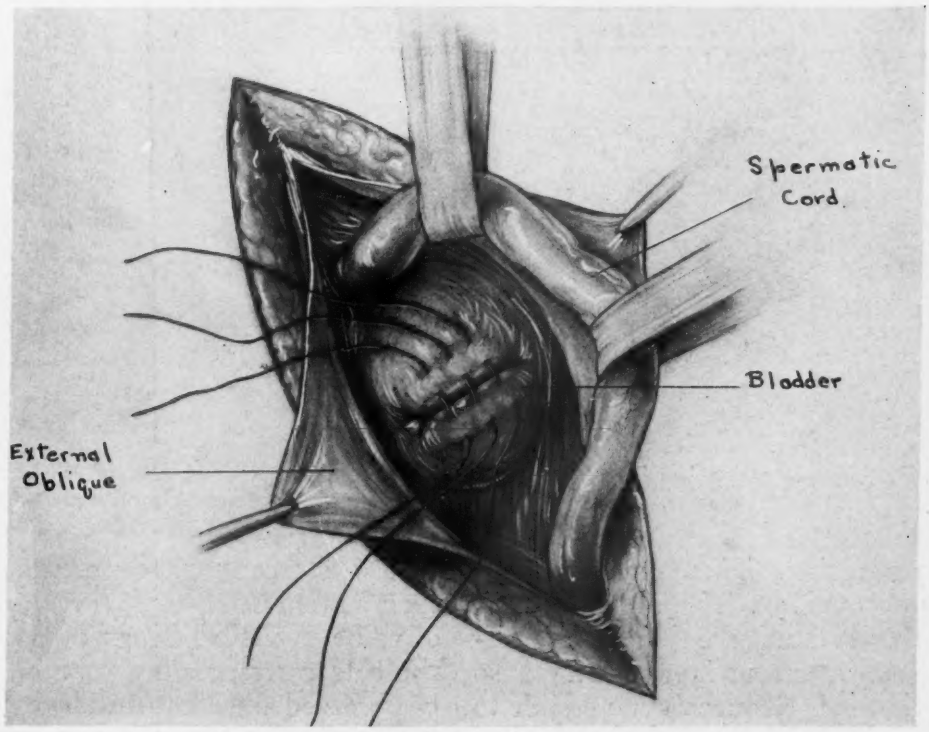

Fig. 8. Reducing redundancy in bladder wall; former method of repairing bladder hernia.

DIAGNOSIS

The diagnosis is not always an easy matter. In fact, it is seldom made presurgically. The condition is strongly presump-

tive if there is a history of a long-standing, partly or wholly irreducible inguinal hernia in a patient past fifty. The mass is usually flat to percussion. There may be an impulse on coughing. The muscular and aponeurotic structures are thin and attenuated. The subcutaneous inguinal ring is so enlarged that it is frequently impossible to determine contour or size.

In incomplete protrusion, the differential diagnosis must rest mainly between herniation of the bladder wall proper through the inguinal (or femoral) ring, and a true vesical diverticulum passing toward the pelvic floor. Routine cystographies are advisable for all patients past fifty years of age with inguinal or femoral hernia.

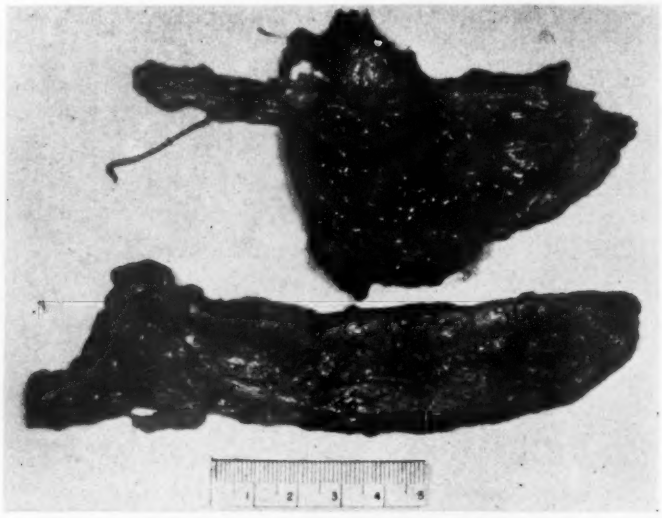

FIG. 9. Exsected portions of a saddle-bag hernia. The upper specimen is a segment of the bladder which formed the medial protrusion through Hesselbach's triangle. The lower one is the lateral part of the hernia, the peritoneal protrusion.

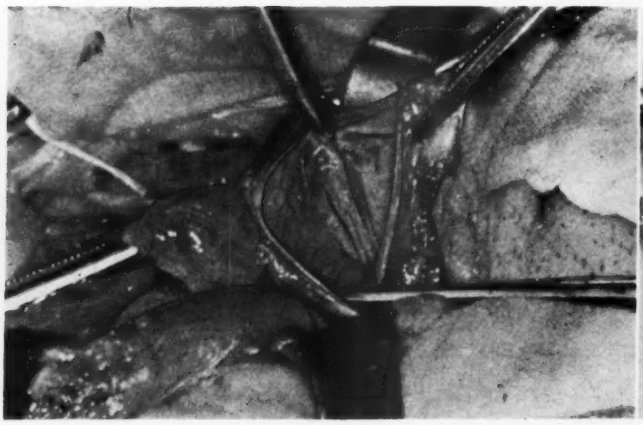

FIG. 10. Bladder protrusion has been resected ready for closure.

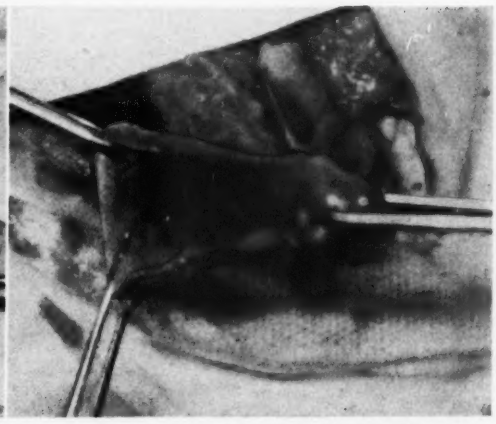

FIG. 11. Another case after resection of bladder protrusion.

A cystoscopic and roentgenographic examination will furnish revealing evidence of value. A vesical defect or protrusion on the side corresponding to the hernia will be observed and confirmed by cystography. When catheterization is resorted to, it will prove to be an important diagnostic aid because the mass will disappear; and if a decrease in the size of the swelling follows the flow of urine, the presumptive diagnosis of bladder herniation becomes a certainty. The phenomenon can be demon-

strated in a reverse manner on injecting fluid or air into the bladder.

TREATMENT

In a recent article I reported eighty cases of pedicle fascia lata flap repair of difficult hernias. I re-operated on the few that recurred and found that the bladder protrusion was responsible for the recurrence which occurred in the medial aspect of the inguinal triangle. I corrected this condition in the manner described below with excellent results.

During the surgical repair it is well to be extremely circumspect regarding: (1) An *undue collection of adipose tissue* and tissue simulating adipose tissue adjacent or contiguous to the hernial protrusion, but commonly medial to the sac. (2) *Isolation of the sac*, which will not, as a rule, prove easy. (3) The *dimensions of the hernia*. It is likely to be extremely large or massive, protruding from preternaturally large rings. (4) *Location*. Urinary bladder hernias ordinarily are found closer to the medial abdominal line than other varieties.

In the past it has been customary with most surgeons to repair the hernia in the accepted manner and to cover the bladder protrusion by reinforcing it with the overlying musculo-aponeurotic structures (Wyllys Andrew, Halsted or Bloodgood technic).

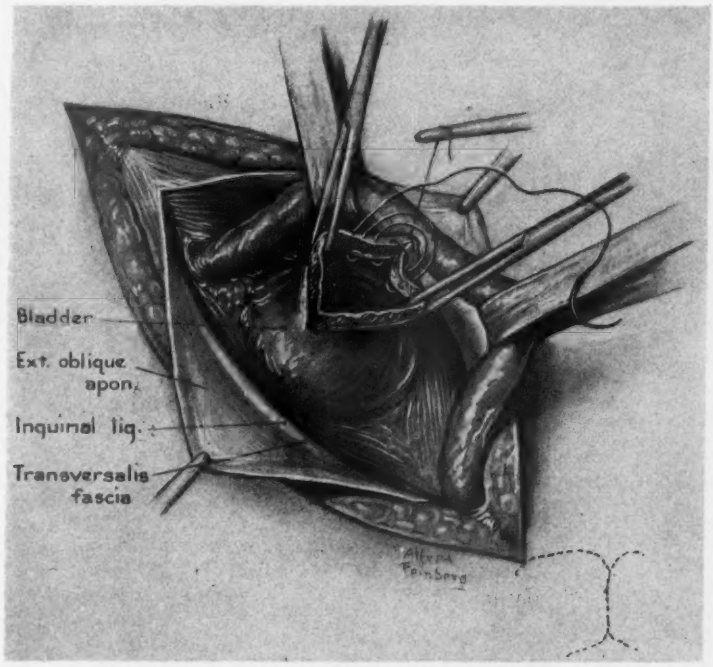

Fig. 12. Closing bladder with Connell suture after excision of herniated part.

I have been in the habit of eliminating the bladder redundancy by either a double purse-string inversion or by a double layer of interrupted Lembert sutures, and then followed the ordinary technic for a direct or indirect inguinal hernia. (Fig. 8.) However, there have been too many recurrences of the bladder protrusion in all of the five varieties previously mentioned. I have, therefore, devised the following procedure:

The repair of the bladder hernia is the same in the five varieties. In the second variety, the small, indirect hernial sac must be removed first, prior to the repair of the bladder protrusion. In this sequence there is no danger of contamination because the bladder has been not only completely emptied by catheterization at least twenty-four hours before the surgical intervention, but irrigated several times with a weak antiseptic solution. The catheter is not

removed until seventy-two hours after the operation. In the third variety, traction is made on the indirect component of the sac (as in the repair of all saddlebag hernias). The bladder protrusion is partly displaced medially; the indirect hernial sac is opened so as to visualize the extent of encroachment on its wall and to avoid puncturing the bladder in the transfixion of the indirect hernial sac. (Fig. 9.) The following technic is then used for the repair of the vesical hernia:

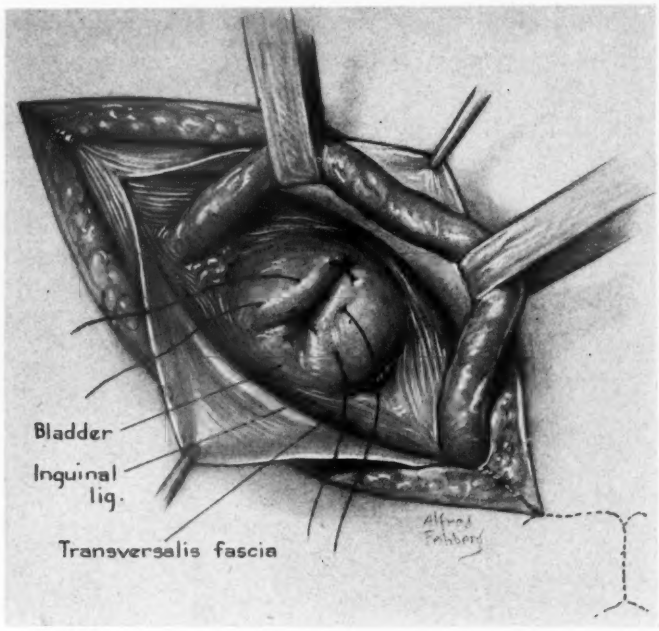

Fig. 13. Re-enforcement with interrupted Lembert sutures.

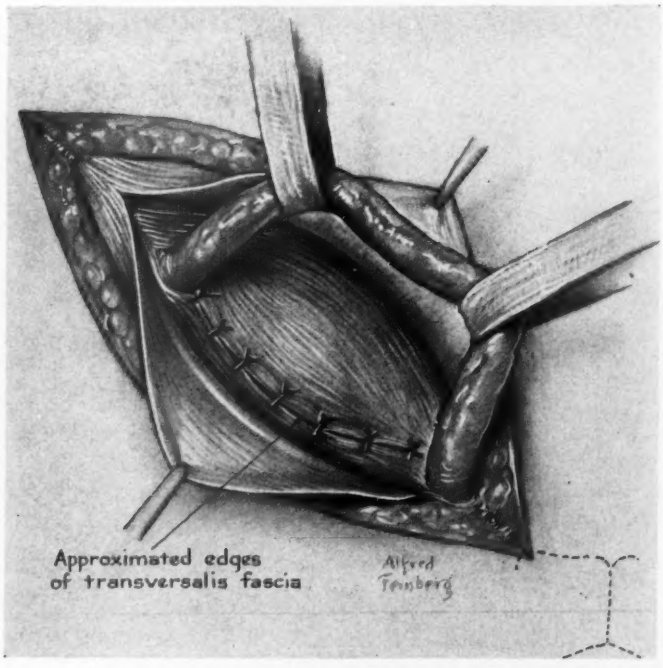

Fig. 14. Approximation of edges of transversalis fascia with interrupted sutures.

The base of the diverticular protrusion is caught by several Allis clamps and the

redundant part exsected. (Figs. 10 and 11.) After establishing hemostasis, the edges of the stoma are inverted by means of a Connell stitch, which is reinforced with interrupted Lembert sutures, utilizing No. 0 chromic catgut on an atraumatic needle. (Figs. 12 and 13.) The margins of the transversalis fascia are then approximated and sutured with interrupted strands of No. 0 chromic catgut. (Fig. 14.) The hernial repair is then continued with the utilization of one of the technics commonly acceptable in the repair of direct inguinal hernia.

Digital exploration of the bladder cavity may be done and closure is then accomplished after a change of gloves.

About 5 Gm. of sulfanilamide powder are insufflated in the wounds before closure of the skin.

The catheter (No. 16 French, soft rubber) remains *in situ* for seventy-two hours. It is sterilized and re-introduced for another twelve hours.

The bladder may be irrigated daily with a warm sterile solution of boric acid followed by instillation of 3 dr. of a 2 per cent solution of mercurochrome.

There have been no recurrences in my series of thirty-seven cases. There was one wound infection, and no urinary extravasation in the surgical area.

Spinal anesthesia was used in all cases.

Table 1

	No.	Average Age	Side	Recurrences One Year Later
Group 1	8	50	5 Right	0
			3 Left	0
Group 2	8	55	5 Right	0
			3 Left	0
Group 3	15	59	9 Right	0
			6 Left	0
Group 4	5	56	5 Right	0
Group 5	1	49	Right	0
Total	37			

One wound infection in Group 3; two cases of mild cystitis in Group 3; one of Group 1 was a female; one case in Group 5 was a female.

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MALIGNANT TUMORS OF THE STOMACH*

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IN a period of four years, from June, 1938, to June, 1942, 127 individuals having malignant gastric tumors were attended on Surgical Section "B" of the Hospital del Salvador in Santiago, Chile. The diagnosis of gastric carcinoma either was made on our service, or the patient had been transferred with this diagnosis from the Medical Section of Professor Alessandri of the same hospital, or from some other hospital or clinic in the Capital or Province.

One hundred four patients (81.88 per cent) were subjected to operation. Fifty eight of these operations (55.76 per cent) resulted in gastric resection. Seven patients were considered inoperable and sixteen refused operation. In 98.55 per cent the microscopic examination showed carcinoma whereas in 1.44 per cent lymphosarcoma occurred.

The following chart reveals the relation of age to gastric cancer, also the distribution according to sex. We found 74 per cent in male, and 26 per cent in female patients.

TABLE I
AGE AND SEX DISTRIBUTION

Age	Female	Male	Total
20 to 30	1	3	4
31 to 40	6	10	16
41 to 50	10	25	35
51 to 60	9	31	40
61 to 70	7	24	31
71 to 80	1	0	1
	34	93	127
Per cent	26	74	100

In the female the greatest frequency of carcinoma is in the fourth and fifth decade. In the male it is in the fifth and sixth decade. That is, in men the disease occurs at a later age than in women.

SYMPTOMATOLOGY

The symptoms which present themselves in patients with gastric cancer are varied, but are of great importance in establishing an early diagnosis and in consequence the institution of radical treatment. The time of evolution, from the onset to the diagnosis on the admission to the hospital, varies from one month to more than two years.

We have classified the symptomatology of our patients and have divided them into five types:

TABLE II
SYMPTOMATOLOGY

	No. Patients	Per Cent	Resections		Inoperable	
			No.	Per Cent	No.	Per Cent
Dyspepsia	73	57.49	35	48.05	28	38.47
Ulcer syndrome	32	25.19	14	43.75	16	50.
Hemorrhage	6	4.74	3	50.	3	50.
Obstruction	9	7.24	3	33.33	6	66.66
Emaciation; asthenia	7	5.51	3	45.	3	45.

These groupings were made according to the symptoms at the onset, but since frequently one type is superimposed on the other or is altered periodically, the patients usually arrive at the hospital with all the symptoms combined.

The most frequent type is the dyspeptic, in more than half of the cases, followed by the ulcerous type in 25 per cent. Hemorrhage as a first symptom occurred in six patients (4.74 per cent) and pyloric or cardiac obstruction in nine (7.24 per cent). Emaciation and asthenia were the initial findings in seven patients (5.51 per cent).

* Address given before the Staff of the Memorial Hospital of New York.

In the evolution of the disease emaciation ultimately was present in 99 per cent of the patients.

In respect to the time interval between the onset of the symptoms and admission to the hospital we have divided the group in the following manner:

TABLE III

RELATION OF OPERABILITY TO DURATION OF SYMPTOMS

	Patients	Per Cent	Inoperable	Per Cent	Operable	Per Cent
1 to 2 months	30	23.62	10	33.33	15	50
4 to 5 months	41	32.28	23	56.09	16	39.02
8 to 11 months	15	11.81	6	40	6	40
1 to 2 years	34	26.77	11	32.35	18	52.93
More than 2 years	7	5.51	3	42.85	3	42.85
	127	.....	53	.....	58 (and 16 refused operation)	

In reviewing the relation between the operability of the tumor and the duration of the disease, we can see that the patients in whom the evolution of symptoms has been present for more than a year possess a greater operative possibility. The explanation of this paradox may be that the cancer is less malignant, in consequence of which evolution is retarded. In the patients who apply for treatment within one to three months of the date of onset, or perhaps his discomforts have caused him to go for an early diagnosis, notwithstanding the more or less rapid growth of his neoplasm, it may be that chance has caused him to arrive at the opportune moment for resection.

TABLE IV

RESULTS OF GASTRIC ANALYSIS

		Per Cent
Anachlorhydria	64 patients	77.10
Hypochlorhydria	14 patients	16.86
Isochlorhydria	4 patients	4.81
Hyperchlorhydria	1 patient	1.20

Anemia of distinct intensity was present in 84.12 per cent there being a normal globular and hemoglobin count in 15.88 per cent.

A history of heredity or possible familial influence was found in twenty-two patients; 15.74 per cent were paternal and 1.57 per cent from other relatives.

Gastric analyses were performed in eighty-three cases with the result shown in Table IV.

X-RAY DIAGNOSIS

X-ray examinations were done on 116 cases; in some of the remaining cases it was not done due to gastric stasis or obstruction or because of established gastroscopic diagnoses or for causes inherent to the service. In these 116 patients gastric cancer was indicated in 109, two were diagnosed as gastric ulcers, one as a degenerated gastric ulcer, two were classified as pyloric stenosis without specification as to cancer and one was diagnosed as a possible benign gastric tumor. In four patients a diagnosis of

TABLE V

RADIOLOGICAL EXAMINATIONS ON 116 PATIENTS WITH GASTRIC CANCER

		Per Cent
Diagnosis of gastric cancer	109	93.96
Diagnosis of gastric ulcers	2	1.72
Diagnosis of degenerated gastric ulcers	1	0.86
Diagnosis of pyloric stenosis	2	1.72
Diagnosis of gastric tumor	1	0.86
Diagnosis of organic pyloric lesion	1	0.86

cancer was not made at the first examination, but was confirmed in the second examination following gastroscopic diagnosis of cancer.

The value of roentgenological examination is of great importance. It permitted the diagnosis of gastric cancer in 96.55 per cent of cases, counting the four diagnoses made after gastroscopic examination. In these "gastric lesions" 85.34 per cent were specifically cancer and in the balance of the cases diagnosis of cancer was confirmed by gastroscopic examination, gastric analysis and the clinical picture.

GASTROSCOPIC DIAGNOSIS

Gastroscopic examination was done on forty-nine of the 127 patients with gastric cancer. The diagnosis of cancer was made in forty-six instances. In thirty-seven patients the diagnosis of associated atrophic gastritis was made which was also confirmed by histological examination. One case was diagnosed as slight superficial gastritis without having been able to visualize the antrum and pylorus where the carcinomatous lesion was; and in one case the diagnosis of polyposis and atrophic gastritis was made. In summary, the diagnostic value of this examination is very good since in our patients the margin of error was only 4.08 per cent and a certain diagnosis was arrived at in 95.92 per cent.

Localization. In determining the location of the gastric cancer in our patients we have taken into consideration the anatomic-pathological examination of the operated patients and autopsies, and the gastroscopic and radiological examinations in the remainder. The most frequent location was on the lesser curvature—29.75 per cent; in the antropyloric region 29 per cent; advanced infiltration of the entire stomach, 19.20 per cent; in the cardiac end 8.26 per cent; in the pylorus exclusively 4.13 per cent; on the greater curvature 1.65 per cent; and in the corpus 2.47 per cent.

Treatment and Results. In the period of four years from June, 1938, to June, 1942, we have treated on our service 127 patients with a diagnosis of gastric cancer with an operability of 81.88 per cent. Taking into account the seven patients considered inoperable and sixteen who refused operation, the resectability was 55.76 per cent including nine total gastrectomies.

The exploratory and palliative operations were performed because of the impossibility of doing a resection due to hepatic metastasis or extensiveness or immobility, due to adhesions of the tumor to other organs or due to its growth into the surrounding structures.

In the following chart we make note of the operations performed, the percentage of operability, the number of deaths in each intervention, and the percentage of deaths.

TABLE VI
OPERABILITY AND MORTALITY RATES

Operation	No.	Operability, Per Cent	Deaths	Mortality, Per Cent
Subtotal gastrectomies	49	47.11	8	16.32
Total gastrectomies	9	8.65	4	44.44
Gastric resections	58	55.76	12	20.68
Gastroenterostomies	12	10.57	3	25.
Exploratory laparotomy	25	24.03	0	0
Jejunostomies	6	5.76	2	33.33
Intestinal resection	2	1.92	1	50.
Gastrostomy	1	0.96	0	0
Operations	104		18	17.30
Refused operation	16			
Inoperable	7			

The total mortality on the service was 15.74 per cent; the postoperative mortality 17.30 per cent.

The operative resectability was 55.76 per cent. This corresponds to 45.7 per cent of all the patients with gastric cancer seen during this period.

Total gastrectomies comprised 15.09 per cent of all the resections for carcinoma. A comparative study of age and mortality of the patients operated upon indicates

that the greater the age the greater the mortality and that operative risks lessen in the young.

TABLE VII
RELATION OF AGE TO MORTALITY RATES

Age	No. of Patients	Death	Mortality, Per Cent
20 to 30	4	0	0
31 to 40	13	2	15.40
41 to 50	24	5	16.67
51 to 60	29	5	17.24
61 to 70	34	7	20.59

The mortality of the gastrectomies is in direct relation to the difficulty of the operation, since the greater the operative risk the greater the mortality. To arrive at this conclusion we have divided the subtotal gastrectomies into difficult and non-difficult cases, taking into account in the first group the difficulty of resection, i.e., adhesions to the surrounding organs, the penetration of the tumor into the intestine and the necessity to resect it, the amplitude of the gastrectomy, the physical unfitness of the patient, etc. These things which lengthen the time of operation diminish the chances of survival.

TABLE VIII
RELATION OF OPERATIVE DIFFICULTY TO MORTALITY RATES

	No.	Deaths	Mortality, Per Cent
Difficult subtotal gastrectomies	17	7	41.17
Non-difficult subtotal gastrectomies	32	1	3.12

The most frequently used anesthetic was ½ per cent novocain local infiltration; on some occasions 1 per cent novocain was added with satisfactory results in most cases; preoperative medication was always used. In some patients unable to stand the operation under local anesthesia, it was supplemented by cyclopropane. We noted that the amount of cyclopropane was less than when used without novocain and afforded better muscular relaxation and less waste of cyclopropane, also less toxic to the patient.

TABLE IX
ANESTHESIAS EMPLOYED

	No.	Percentage
Gastrectomies		
Local novocain	20	37.04
Local and cyclopropane	10	18.52
Spinal procain	9	16.67
Cyclopropane	7	12.96
Local and anterior splanchnic	4	7.40
Cyclopropane and ether	3	5.65
Ether	1	1.85
Exploratory Laparotomies		
Local novocain	13	52
Local and cyclopropane	2	8
Spinal procain	3	12
Cyclopropane	5	20
Local and anterior splanchnic	2	8
Gastroenterostomies		
Local novocain	7	58.33
Local and cyclopropane	2	16.67
Spinal procain	1	8.33
Cyclopropane	2	16.67
Jejunostomies		
Local novocain	5	83.33
Spinal procain	1	16.67
Gastrostomies		
Local novocain	1	100
Exclusion Resection		
Local novocain	1	100

The most frequent postoperative complications are of respiratory origin ranging from a simple bronchitis to pneumonia and bronchopneumonia. Lately respiratory complications have diminished due to the

use of the sulfa drugs which were used at first as a curative measure and then as a preventative, beginning its use twenty-four to forty-eight hours before operation and continuing for from three to four days postoperatively. The drug is discontinued when the temperature is no longer elevated.

TABLE X
POSTOPERATIVE COMPLICATIONS

	No.	Percentage
Gastrectomies		
Respiratory	9	40.90
Infections of the wound	4	18.18
Cardiac	2	9.09
Peritonitis	2	9.09
Shock operative	2	9.09
Jaundice	2	9.09
Gastric fistula	1	4.54
Postoperative complications in gastrectomies	22	37.93
Exploratory Laparotomy		
Respiratory	2	66.66
Thrombophlebitis of lower limbs	1	33.33
Complications postexploratory laparotomy	3	12.
Gastroenterostomy		
Respiratory	1	33.33
Diarrhea	1	33.33
Shock	1	33.33
Postoperative complications in gastroenterostomies	3	25
Jejunostomy		
Respiratory	1	100
Postoperative complications in jejunostomies	1	16.66
Exclusion Resections		
Respiratory	1	100
Postoperative complications in exclusion resections	1	50

The same medication diminishes the number of wound infections, also the frequency of peritonitis. In the last year postoperative pneumonia has practically disappeared. We had only one case in which the sulfa drug was omitted, which corroborates the necessity for its routine use in patients with gastric cancer who are scheduled for the operating room.

HISTOPATHOLOGICAL EXAMINATION

The histological examinations performed in the service of Surgical Pathology of this hospital gave the following results:

TABLE XI
HISTOLOGICAL TYPES

	No.	Percentage
Adenocarcinoma	37	53.62
Mixed cell carcinoma	12	17.39
Solid carcinoma	12	17.39
Gelatinous carcinoma	3	4.34
Schirrhous carcinoma	2	2.89
Degenerated ulcers (adenocarcinoma)	2	2.89
Lymphosarcoma	1	1.44

The pathological examination is of great importance in recognizing the histological type and the evidence of metastasis in the lymph nodes since this gives us the prognosis, because if it is a sarcoma we know that radiotherapy is indicated and that if there are no lymphatic metastases in a carcinoma the prognosis is more favorable. For this reason we have divided the distinct histological types with respect to lymphatic metastases in Table XII.

According to the Borrmann classification the results are as shown in Table XIII.

We find the greatest frequency in carcinoma in Type II Borrmann followed by Type IV, having the lowest percentage in Type I.

Of the inoperable carcinoma in which a histological examination by biopsy was done we found eleven adenocarcinomas, one solid carcinoma, one mixed cell carcinoma.

TABLE XII

RELATION OF HISTOLOGICAL TYPE TO LYMPHATIC METASTASES

Without Lymph Node Metastasis	With Lymph Node Metastasis	Examination Did Not Specify
Adenocarcinoma and catarrhal lymphadenitis..... 6	Adenosarcoma metastasis in lymph node..... 24	Adenocarcinoma..... 7
Mixed cell carcinoma and catarrhal lymphadenitis..... 1	Mixed cell carcinoma and lymphatic metastasis..... 9	Mixed cell carcinoma..... 2
Solid carcinoma & catarrhal lymphadenitis..... 2	Solid carcinoma and metastasis to lymph nodes..... 9	Solid carcinoma..... 1
Gelatinous carcinoma and catarrhal lymphadenitis..... 1	Gelatinous carcinoma and metastasis to lymph nodes..... 1	Gelatinous carcinoma..... 1
Scirrhous carcinoma and catarrhal lymphadenitis..... 1	Scirrhous carcinoma and metastasis to lymph nodes..... 1	
Degenerated ulcer (Adenocarcinoma) and lymphadenitis..... 1	Degenerated ulcer (adenocarcinoma) and lymphatic metastasis..... 1	
12 cases with catarrhal lymphadenitis..... 21.05 per cent	45 cases with lymph node metastasis.. 78.94 per cent	

The relation between the duration of symptoms and the histological type are as shown in Table XIV.

TABLE XIII

BORRMANN CLASSIFICATION

Group	No.	Percentage
I	3	5.88
II	19	37.25
III	11	20.52
IV	18	35.29

By the preceding picture we can deduct that the mixed cell carcinoma is slower of evolution because the majority of patients arrive at the hospital later; adenocarcinoma follows, solid carcinoma being the most rapid in progress. Hence the majority of these patients arrive before three months have elapsed since the onset of symptoms. What has been said previously confirms the relation of the time between the onset in inoperable carcinoma and the histological examination.

We see that the inoperable carcinoma with the shortest time of development was the solid, followed by the adenocarcinoma, the mixed being the slowest.

PROGNOSIS

We consider the prognosis in those individuals who did not die postoperatively allowing a postoperative period of thirty days. This prognosis varies with each intervention for which reason we shall view them separately. The greatest difficulty in the region of our hospital is the control of postoperative follow-up. Of our total patients, 36.43 per cent of those

TABLE XIV

RELATION OF HISTOLOGICAL TYPES TO DURATION OF SYMPTOMS IN OPERABLE CARCINOMAS

Duration of Pain	Adenocarcinoma	Solid	Mixed	Gelatinous	Scirrhous	Lymphosarcoma
1 to 3 months	9	6	1	..	1	1
4 to 6 months	11	3	2			
7 to 12 months	6	2	5	2	1	
13 to 24 months	6	1	1	1		
More than 24 months	2	1	3			

treated on this service in four years have kept us informed as to their state of health. Of the gastric resections we have been able to do a postoperative follow-up on 55.17 per cent.

TABLE XV
RELATION OF HISTOLOGICAL TYPE TO DURATION OF SYMPTOMS IN INOPERABLE CARCINOMA

Duration	Adeno-carcinoma	Solid	Mixed
1 to 3 months	..	1	
4 to 6 months	7		
7 to 12 months	4		
More than 12 months	..	..	1

The patients who had total gastrectomies had an average postoperative life of six months. Only one is alive and in good condition fourteen months after the operation.

The subtotal gastrectomies have an average life of seven and one-half months, aside from those who actually live amounting to 28.57 per cent.

More than 4 years	1 patient
More than 2 years	2 patients
More than one year	2 patients
6 to 11 months	4 patients
1 to 6 months	5 patients

Average survival	Gastroenterostomies	2½ months
Average survival	Exploratory laparotomies	4 months
Average survival	Gastrostomies	2 months
Average survival	Jejunostomy	1 month
Average survival	Inoperable	2 months

The longest survivals in patients with gastric cancer, aside from the resections, were those who underwent exploratory laparotomy. They averaged four months of postoperative life while the palliative operations such as gastroenterostomy, gastrostomy, jejunostomy, etc., averaged two months. Therefore, in gastric cancer if it is not possible to perform a radical operation such as a gastrectomy only an exploratory laparotomy should be done since it is the only one that prolongs life.

The gastrectomies with an average survival of seven and one-half months showed the existence of lymph node metastases in all but two cases which indicated catarrhal lymphadenitis.

Of the patients who had gastrectomy for cancer and who actually survive with a diagnosis of only catarrhal lymphadenitis in the perigastric one has lived four years, one has lived two years, and one is living two months later. In the remainder the histological examination showed metastasis to regional lymph nodes.

RELATIONSHIP OF THE OPERABILITY OF GASTRIC CANCER TO THE INTEGRITY OF THE HEART AND VASCULAR SYSTEM

Out of the 127 patients treated for gastric cancer four had cardiac lesions with cardiac insufficiency which, under treatment, became compensated prior to the operation. The operation was then performed without difficulty. Two days before the operation all of these patients received ¼ mg. of estrofantina daily which treatment was continued for three or four days postoperatively. In the following chart we make note of the different diagnoses, operations, anesthesias employed, and the prognosis.

BENEFIT OF GASTRECTOMY IN PATIENTS HAVING GASTRIC CANCER WITH AN AVERAGE LIFE OF SEVEN MONTHS

Of the patients who died several months after a gastrectomy, eighteen continued to have symptoms of their illness which did not permit the patients to resume their normal activity, or 50 per cent. The other 50 per cent were able to resume an active life although in some cases for as short a period as one to two months, and others for one year until the symptoms returned and the patient died. Two patients (11.11 per cent) returned to their activities and died from other causes than gastric cancer, cardiac insufficiency having caused their deaths.

SUMMARY

1. In the four years that we have been attending 127 patients with gastric cancer,

84 per cent of the total were seen in the last two years, due to the recognition of the specialization of this matter on this service.

TABLE XVI

GASTRECTOMY IN PATIENTS WITH HEART DISEASE

No. of Observation	Age	Diagnosis	Operation	Anesthetic	Prognosis
37—1950	65-A	Gastric cancer—aortic insufficiency; arteriosclerosis	Subtotal gastrectomy	Local novocain	Died, 8th day, peritonitis
39—8721	58-A	Gastric cancer; hypertension; low pulse pressure; rheumatic mitral insufficiency	Subtotal gastrectomy	Local novocain	Died, two months. No metastases; cardiac insufficiency
39—5521	41-A	Gastric cancer—aortitis—aortic insufficiency (Hodson)	Subtotal gastrectomy	Local novocain	Died, 4 months; cardiac insufficiency; no metastases
40—8471	55-A	Gastric cancer; aortic insufficiency; luetic aortitis	Total gastrectomy	Local splanchnic	Died, 7 months; right pleural metastases; carcinomatous peritonitis

2. We have observed the greater frequency of gastric cancer in the masculine sex (74 per cent) and in the fifth decade of life.

3. The presence of dyspeptic symptoms (57 per cent) or the ulcer syndrome (25 per cent) in individuals over forty years and without previous digestive disturbance should lead us to suspect gastric cancer.

4. The gastric resectability is greater (52 per cent) in individuals whose symptoms are of one year's duration than in those patients with shorter histories, since they are tumors of slow evolution.

5. The lesser curvature and prepylorus are the regions in which gastric cancer localizes more frequently. They interfere with the flow of gastric juice, producing anachlorhydria or else the cancer spreads itself into previously degenerated mucosa (chronic gastritis).

6. Gastric resectability is one of the highest of those published (55.76 per cent) with a mortality of 20 per cent corresponding to a 16 per cent in subtotal gastrectomies and 44 per cent in total gastrectomies.

7. The operative mortality increases in direct relation with the age and the difficulty of the operation.

8. The most frequent postoperative complications are: respiratory and wound infections, whose percentage has diminished notably since the routine use of the sulfanilamides.

9. The evolution of gastric cancer is in possible relation with its histological type; the slowest evolution has been the mixed cell cancers followed by adenocarcinoma. The solid cancers are the most rapid in growth.

10. The prognosis is better for exploratory laparotomy than for the palliative operations, i.e., gastroenterostomy, jejunostomy, gastrostomy, etc.

11. The gastrectomies were executed without difficulty in four patients with severe cardiac difficulties, one of which was a total gastrectomy with equal good evolution postoperatively, as easily as in an individual with normal cardiac function.

12. The only treatment in gastric cancer is radical gastrectomy performed at an opportune time.

SUBMUSCLE PELVIC TISSUE SPACES

ANATOMY AND CLINICAL CONSIDERATIONS

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THE anatomy of fascial spaces, muscles, the retroperitoneal space and the pelvis in general is a "bug-bear" for the medical student and this dilemma is not clarified in the mind of the practitioner as the years separate him from the dissecting room. It is the author's belief that this general hazy conception of these structures is due to the lack of a clear knowledge of the embryological development of these areas, plus a lack of correlation of this knowledge with parts such as the anterior abdominal wall where the anatomy is well understood. This paper is written to show this correlation and to give some surgical applications of the same.

The ventral and caudal ends of the embryo are formed during the first two weeks of fetal life. At the time of formation of the embryonic disc the three primitive tissue layers are present, i.e., ectoderm, mesoderm and entoderm. From the embryonic disc all the bodily structures are formed. Growth proceeds from this flat structure in all directions. Some parts show more rapid growth than others; there are out-growths and in-growths here and there, but in general there is an outward and downward growth meeting in the midline ventrally to form the abdominal wall. This general plan may best be visualized by comparing the embryo at this early stage to a sealed envelope. The address side represents the embryonic disc area with growth extending out and down in all directions, the ventral part coming together by four flaps, one at each end and one on each side. The side flaps may be considered to form the abdominal wall. The cephalic flap is not very distinct in the fully developed embryo but may be considered, for analogy at least, to be represented by the fascial and superficial neck muscle, the pectoralis muscles and the rectae muscles of the abdominal wall. The caudal flap forms the caudal or rectogenital end of the body. The clear conception of this picture of development is essential for the correlation and remembering of the pelvic structures in their proper relationship.

These flaps grow in all directions and are made up of their three primitive germ layers. Nothing will be said about the ectoderm as this becomes visible and easily understood. The mesoderm forms the subcutaneous tissue, fat, muscles and fascia. On the abdominal wall this layer is represented by subcutaneous tissue, external and internal oblique and the transversalis muscles, the submuscular layer of loose areolar tissue known as the transversalis fascia and peritoneum. This later will be referred to hereafter as the submuscle tissue layer. For simplicity and clarity the above three muscle layers will be considered as one mass. This gives five definite layers, i.e., skin, subcutaneous tissue, muscle, submuscle tissue layer and peritoneum. It should be noted here that the submuscle tissue layer extends posteriorly to the spinal column. It is within this tissue that the retroperitoneal structures such as pancreas, kidneys and ureters lie. Garota's fascia is a specialized portion of this tissue enveloping the kidneys.

The same layers are present in the caudal flap. Here the muscle layer is represented by the levator ani, compressor urethrae

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and the muscles in the skin and subcutaneous tissue, i.e., dartos, perineal and the sphincter muscle. It is these muscles, especially the former, that cause so much distress and confusion in the mind of the student and physician.

Phylogenetically, the levator ani muscle has served several functions. In the fish and salamander group of animals this muscle flexes the tail producing the main means of locomotion and protection. In the mammals, such as the cow and the horse, the levator ani is a fairly strong muscle and flexes the tail from side to side principally for protection against insects. In man this muscle losses its tail swinging properties. Instead by a more direct ventral attachment to the pubis and the sacrum it forms a sling-like structure which on contracting attempts to shorten the distance between these two immovable points thereby forming a solid band or floor for the pelvis. The rectum and urogenital sinus pierce this muscle in the midline forming potentially two round orifices. By attachment at each end and to a central point, the superficial perineal muscle forms a figure-of-eight about these orifices external to the levator ani muscle. This arrangement produces efficient sphincters for both orifices.

Below or internally to the levator muscle, as with the abdominal wall muscle mass, is the submuscle areolar tissue known as the urorectal or urogenitorectal fascia. This fascia is continuous with that over the abdominal wall and occupies a space which in this paper is called the submuscle tissue space. It is spoken of clinically as the retroperitoneal space. Actually there is no space here, this space being taken up by areolar tissue, fat nerves, blood and lymph channels.

This submuscle tissue layer is so important and difficult to comprehend that a more detailed consideration will be given. For this it is desirable to return to the study of the embryo for clarification.

In the 2 mm. embryo the primitive gut lined by entoderm is made up of the yolk sac. It is hardly a sac and had better be considered a flat mass of cells extending downward from the embryonic disc to meet in the midline where the four envelope-like flaps meet ventrally. Covering this structure is a mass of unidentified cells, the mesoderm. This mass of tissue divides into two major parts, i.e., the outer or muscle forming layers, referred to above, and the inner fascial layer now under discussion. The cell layer bordering on the primitive gut tract forms two layers with a cavity in between the future abdominal cavity, the cells forming the visceral and parietal peritoneum. The primitive gut settles ventrally pulling the peritoneum and the submuscle fascia with it forming the mesentery. It can be seen that the submuscle layer acts as a buffer or cushion between the peritoneum, both visceral and parietal, and the muscle layer.

Early this mesodermic mass has a considerable thickness. As growth proceeds it becomes compressed to a thin tissue, at least the inner portion, in places becoming little more than a potential space. If the student will retain the conception of the gut tract and mesentery as being a flat partition-like structure hanging from the midline and give the inner portion of the mesodermal mass the relative thickness it once had in embryo, he can see it as an organ of considerable size, easy to trace and comprehend. Besides forming the skeletal tissues this mass of cells form the blood vessels, lymph nodes and vessels, the urogenital system and the supporting structures of these tissues. Normally, these structures seemingly do not occupy space but in disease such as inflammatory swellings, abscess ramifications, or neoplasms there is an attempt of varying degrees to restore thickness to this tissue comparable to that of the embryo. For a sound surgical attack on these disease conditions, the surgeon should have the anatomy of this submuscle tissue layer well in mind.

SURGICAL CONSIDERATIONS

Superficial Infections. A carbuncle or abscess located in the tissue external to the muscles of the abdominal wall usually gives

the surgeon little doubt as to the location of pus and the place of drainage. The depth and ramifications of an abscess in the perineum is not so well understood by the profession in general. Viewed as a part of or similar to the abdominal wall, the anatomy becomes clarified. The much discussed ischiorectal fossa is only a diamond shaped subcutaneous space filled with fat and pierced by the rectum and urogenital tract. The deep wall is formed by the levator ani muscle. Doctor Batson found by actual measurement that this fossa could hold no more than four ounces on a side in the average adult. When an abscess in this area is opened and more than four ounces of pus are obtained it can be assumed that some of the pus is draining from an abscess on the opposite side or deep to the levator ani muscle, i.e., in the submuscle tissue space and that adequate provision should be made for drainage of these areas.

Deep Infections. By the term "deep infections" is meant infections deep to the muscle layer. It is the infection of the submuscle tissue layer. It includes such well known entities as retrocecal abscess, subphrenic abscess, abscess of the broad ligament, perinephritic abscess, periprostatic abscess, pelvic cellulitis following trauma and infection such as that following child birth, urinary extravisation from extraperitoneal rupture of the bladder, infection of the space of Retzius and the infiltrating and fibrosing chronic diseases such as lymphopathia venerum. By reviewing the ramifications of the inner portion of the embryonic mesoderm layer one can see that all these clinical entities are infections or abscess in one structure, the names only specifying in what area the disease is located. The extension of inflammation or abscess from one of these areas to the other can be readily understood. By the same review the surgeon knows he must pass through the skin, subcutaneous tissue and a muscle layer and that only, before he can institute drainage.

Certain technics such as the transpleural posterior approach to a subdiaphragmatic abscess is a departure from the above anatomic simplicity. Abscess, cellulitis or urinary extravisation about the base of the bladder may be drained by an approach to the submuscle tissue space suprapubically, i.e., through the space of Retzius or better through the central part of the perineum, traversing through or between the levator ani muscle into the infected region. Opening a broad ligament abscess through the vagina is essentially the same route as given above only taking advantage of a natural orifice. Another approach to the broad ligament abscess is at the level of the internal ring, following the round ligament retroperitoneally and down into the true pelvis. If desired through-and-through drainage may be instituted from the internal ring to the vagina. Likewise the space of Retzius may be drained suprapubically and/or through an incision in the perineum.

It is not uncommon to find varying amounts of red and white cells in the urine in acute appendicitis. These cases are usually retrocecal appendices and the disorder is appendicitis, periappendicitis, infection of the submuscle tissue or retroperitoneal space, peri ureteritis and ureteritis.

There has been much conjecture and debate on how bacteria from the intestinal tract get from that organ to produce such diseases as pyelitis, cholecystitis, cystitis and other abdominal conditions. If one views the submuscle space as one organ connected throughout by a network of lymph vessels, one can easily understand how once the bacteria have gone through the intestinal wall they might go directly or by reflux to any of the organs in or bordering on this tissue space.

Probably a large number of the so-called referred pain syndromes in and about the abdominal organs could be explained on a more exact pathological basis if one considers that all the visceral nerves and part of the peripheral nerves traverse this submuscle tissue. Lymph drainage from one

diseased area can produce a lymphangitis some distance from the site of the original infection; this in turn produces perineuritis and neuritis and from these give rise to what is termed referred pain. Such conditions probably explain some of the confusing symptoms encountered in diseases of the pelvic organs.

Carcinoma and lymphopathia veneria spread along the lymph channels. In general the lymph channels follow along the blood vessels supplying the organs. One can readily see how these diseases by infiltration and pressure cause obstructive uropathy and stricture of the rectum. To obtain relief from the obstruction the opening in the ureter or bowel must be proximal to the diseased region where the blood and lymph vessels course through the submuscle tissue space.

Pelvic Repair. Many gynecologists, upon being asked what tissues they use and depend upon for the support and correction of cystocele and rectocele, reply that they suture together the pelvic fascia. On further questioning they cannot give a clear description of this fascia nor from what it was derived. Those who depend upon the "pelvic fascia" to correct cystoceles and rectoceles permanently are doomed to some disappointment.

The submuscle tissue which occupies the submuscle tissue space in general is a loose areolar tissue having varying amounts of fat depending upon the individual and place of the body. In the pelvis it folds over in areas to form rather weak ligaments and fascia, i.e., the uterosacral, the broad ligament, the uterovesicle ligament in the female, the prostatic capsule and subvesicle ligament in the male, and the so-called pelvic fascia in both sexes. The support of the pelvis and pelvic structures is primarily performed by the levator ani muscle just as the abdominal muscles are the chief support for the anterior abdominal wall. Therefore, in the correction of cystocele and rectocele, the surgeon's prime aim should be to restore to normal the sling-like plane of the levator ani muscle reducing the orifice of the vagina and the rectum to as near slit-like openings as is possible and yet feasible for good function. The figure-of-eight configuration of the superficial muscles about the rectum and vagina should be remembered when an attempt is made to restore this crossed over insertion, when destroyed, in order to restore normal sphincter action.

SUMMARY

An effort has been made to correlate the easily understood anatomy of the anterior abdominal wall with the poorly understood anatomy of the pelvis and retroperitoneal space. Once this correlation is understood the student or physician can easily and quickly orient the different layers. Some surgical applications of this knowledge have been presented. As the internist must know his drugs, so must the surgeon know his fascial spaces.

APPENDICITIS*

A REVIEW OF 4,283 CASES

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THIS paper is based on the experience gained from a series of 4,283 appendectomies compiled from personal office and hospital records. The cases comprise appendectomies performed in the Jewish and Mount Sinai Hospitals from 1913 to January 1, 1942. The series includes operations on the appendix for acute, subacute and chronic lesions. In the above total are included appendectomies done in the course of abdominal operations performed for other conditions.

CHRONIC APPENDICITIS

There is no question that chronic appendicitis is a distinct clinical entity. Absolute cure of many patients follows the removal of an appendix packed with foreign bodies of all kinds. Fecal concretions constituted the large majority of abnormal bodies found in the appendix, pin worms were not uncommon, seeds, round worms and solid objects were rarely found. Two cases of carcinoid of the appendix were uncovered, as a result of symptoms resembling those of chronic appendicitis. Especially annoying are the symptoms referable to those appendices situated retrocecally and bound to the posterior wall of the cecum. The u-shaped appendix, so formed by adhesions which bind down the mid-portion of the viscus commonly results in nagging symptoms. Its removal may cause relief from complaint that is often spectacular. Symptoms of chronic appendicitis may occur in children and adults. In children the symptoms are usually due to congenital malposition of the appendix dependent upon non-rotation of the cecum placing the appendix in a retrocecal position.

INCIDENTAL REMOVAL OF APPENDIX

The incidental removal of the appendix should almost always be undertaken while operating for other abdominal conditions. I have never had to regret this additional step even in the presence of a ruptured extra-uterine pregnancy. The appendix should be removed when the primary operation is a cholecystectomy except in rare instances.

ACUTE APPENDICITIS

When the classical symptoms of pain, tenderness and rigidity in the right lower quadrant are present, the diagnosis should not be difficult. There are, however, many variations from this picture which frequently tax one's ability to make a proper diagnosis. In many cases there is a variable amount of discomfort depending upon the stages of the disease. Rigidity may be very marked without much inflammation of the appendix at the time of operation. Conversely, rigidity of the abdomen is often slight especially in fat individuals in whom the appendix is commonly gangrenous. The physical examination is a most reliable means of making a diagnosis. The white blood count, particularly the Schilling modification is of some assistance, but greater reliance must be placed upon the degree of rigidity present. This is frequently difficult to evaluate and accurate diagnosis often depends on a "sixth sense" which is acquired only after one has palpated hundreds of abdomens. The blood

* From the Services of the Jewish and Mount Sinai Hospitals.

count is undoubtedly helpful, but should be used only as an adjunct in obtaining a diagnosis. The manifestations of appendicitis in children and adults varies. Appendicitis in children frequently does not follow the rules. Rigidity and tenderness are often difficult to elicit especially in very young children. In children up to five or six years of age it is usually most difficult to obtain proper data for arriving at a conclusion as to the cause of the illness. The symptoms in children are rather variable and little dependence can be placed upon the child's statements. But the greatest dependence must be placed upon the abdominal examination by the physician. Drawing up of the leg during palpation of the right iliac fossa is the most important sign of an inflamed appendix. The physical examination of the abdomen supersedes the laboratory examination. The stage of the disease, the anatomical location and the type of incision can be determined by careful evaluation of all the factors before operation.

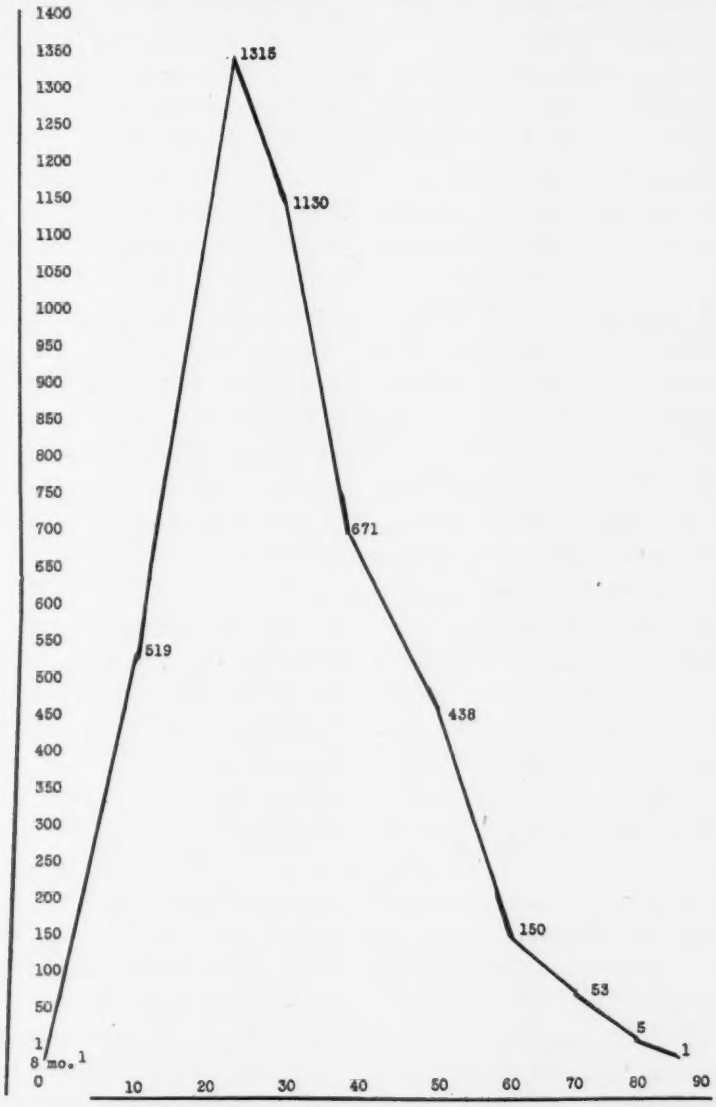

Fig. 1. Chart showing the age range in 4,283 cases of appendicitis.

AGE

The youngest patient operated upon was a baby eight months old. The oldest patient operated on was eighty-one years old. The former recovered as did the latter.

It will be noted (Fig. 1) that the greatest number of patients (2,445) were operated upon between the second and third decades. In the fourth and fifth decades there were 1,109 patients operated on.

As stated above appendicitis in children progresses rapidly and no time should be lost operating upon them. One child eighteen months old was sick three days with symptoms of appendicitis. At operation a perforated appendix with peritonitis was found. He recovered.

Appendicitis in the aged is a serious condition because the great majority are far advanced cases, usually of the gangrenous perforated type.

CAUSE

The cause of appendicitis is unknown. Hematogenous infection plays a part undoubtedly in many cases, and is particularly dangerous when associated with "sore throat." Obstruction to the circulation may be due to fecoliths, other foreign bodies as well as to anatomical deformities. Indiscretion in diet is often a determining factor. I have noticed, however, a strong relationship between the eating of "hot dogs" and appendicitis. John B. Deaver often stated in his clinic that an appendix once infected is always diseased. He also said "the appendix never forgets," meaning of course after the first attack others are sure to follow. However, obstruction of the appendiceal lumen from without or from within appears to be the trigger which sets off most acute attacks.

INCISIONS

Many types of incisions have been advocated: the straight incision, the McBurney and the so-called extraperitoneal or lateral approach for those appendices lying in the loin space. Much has been written about the use of the McBurney incision in acute appendicitis. I use it in some types of this disease, especially in those suffering from chronic appendicitis. In my opinion, it is a fallacy to state that the mortality of acute appendicitis can be reduced by the use of the McBurney incision. The field through which one works is very limited and, therefore, in removing an appendix acutely inflamed more traumatism is inflicted. It is also claimed for the McBurney incision that when an infection occurs in the wound there is less likelihood of hernia resulting. I have observed this complication following infection even after the muscle splitting incision. With the straight incision, the site of operation can be visualized more thoroughly and the appendix, therefore, can be removed more readily. It is a well known fact that the location of the appendix anatomically may vary. It may assume twelve to fourteen different positions. The incision should always be planned with this fact in mind.

SULFA DRUGS

The indications for the use of sulfanilamide and other sulfa drugs have been materially broadened on account of its use in inflammatory conditions other than streptococcic infections. It seems to do more good in those cases that have perforated causing as a result a localized abscess or a spreading peritonitis. In some patients the results are magical. Even before the use of sulfanilamide the majority of these cases recovered but there is no doubt the sulfa drugs have saved many lives. Since 1937, when prontosil and its derivatives were introduced we have encountered four deaths. A child eight years old was admitted to the hospital in February, 1937, with symptoms of general peritonitis. Upon opening the abdomen a large amount of pus exuded. The appendix was removed but was not considered the cause of the peritonitis. Prontosil was used. One patient was admitted to the medical wards in February, 1940. He was transferred to surgery after several days of observation. At operation a suppurative appendicitis was found. He developed later a pelvic abscess which was drained. Following this he eventrated the contents of the abdomen. Sulfanilamide was used. A patient

sixty-four years old was admitted to the hospital, March, 1941, several days after the initial symptoms of appendicitis. When operated upon an acute perforated gangrenous appendix accompanied by general peritonitis was found. Sulfanilamide was used without avail. A child three years old was admitted to the hospital, October, 1941. At operation an acute perforated appendix was found. The patient received 8 Gm. of sulfanilamide after operation on account of a misunderstanding between the medical and surgical services.

COMMENT

While the sulfa products have acted magically in many cases, the impossible cannot be expected in a review of the above reported deaths. In the first case we were dealing with a primary peritonitis, a rather fatal disease in children. In the second case, consultation was not asked for at the proper time. The third case was treated too long at home. In the fourth case it is evident an overdose of sulfa was administered.

EMERGENCY OPERATION VERSUS DELAYED OPERATION

There is no question that the diagnosis once having been made, the appendix should be removed immediately. In children the operation should be performed at any time of the day or night because in the young the appendix has the peculiar faculty of becoming gangrenous in a comparatively short time. The absence of pain after a very severe initial manifestation portends great danger, especially in children. I have been consulting surgeon to the Foster Home for Jewish children for over twenty-five years. By making the resident nurse appendicitis conscious, abdominal pain in a child is promptly reported. We have never lost a patient following operation for this disease at this institution. In adults after the symptoms have been present for two or more days it may not be necessary to operate immediately especially if they arrive in the wee hours of the night. It is better to give glucose parenterally and remove the appendix at an opportune time in the morning. An inflamed appendix should always be removed. Simple drainage of the abscess is an incomplete operation. A gangrenous appendix that remains is always a source of infection and the sinus commonly does not heal unless the offending organ is finally removed. There are some neglected cases encountered where the appendix has entirely sloughed off at its base. In these cases nature has amputated the appendix. Drainage of the abscess is the preferred operation in such instances. Many of these cases require a subsequent operation for the closure of a fecal fistula. A gangrenous, perforated appendix adjacent to its insertion at the cecum requires a special technic. A cecostomy should be performed and a pezzer catheter inserted, protected by the great omentum. Obviously inversion of the cecum cannot be done.

DRAINAGE

With a greater experience the surgeon is inclined to use less drainage after operation for acute cases. In a patient, however, where perforation has occurred and a localized collection of pus is found, drainage should be instituted. The best results are obtained in badly infected cases when the wounds are kept wide open. All drainage is removed at the end of five days. When stitches are considered necessary because of a lengthy incision, through-and-through silkworm gut or dermal sutures are passed through all the layers of the abdomen and loosely tied. No attempt is made at tier suturing. The incidence of hernia is not increased by through-and-through sutures. In fact it has always seemed to me the danger was less with this technic.

ANESTHESIA

As a result of the division of appendicitis into acute, subacute and chronic types, the kind of anesthetic used can also be varied depending upon the degree of inflammation

that exists. In chronic appendicitis a general anesthetic may be used. In patients in the acute stages of the disease, those in whom we suspect perforation, localized peritonitis, abscesses or an acute unruptured appendix with high leukocytosis, spinal anesthesia is the choice as compared with all other types of anesthetics. As stated in a former paper, spinal anesthesia saves lives rather than destroys them. On account of the relaxation of the abdominal wall one can localize the operating area without spreading the infection. This is probably the most important factor in the reduction of mortality in patients suffering from acute perforated appendicitis. As a result of this type of anesthetic I have used what I have been accustomed to call the two forceps technic. In this technic the colon is visualized through the incision and two Russian tissue forceps are used, one in either hand to pick up the cecum. This frequently allows the appendix to be delivered into the wound without the insertion of the finger into the abdominal cavity. In children ether is the anesthetic of choice. On account of an upper respiratory infection chloroform was administered to a child twelve years old. She died of acute yellow atrophy of the liver which was verified at postmortem examination.

Complications depend largely upon the care and the gentleness used at the time of operation. Some complications are unavoidable. Intestinal obstruction, for instance, may result from a simple adhesion between the intestine and the parietal peritoneum or from an adhesive kinking of the gut upon itself. Symptoms of intestinal obstruction may appear shortly after operation and the sooner these adhesions are released and the intestines placed in their normal position again the more quickly will the symptoms subside. I have performed five successful operations all in a comparatively short time on the same patient with persistent intestinal obstruction. These operations consisted in the release of adhesions, ileostomy or colostomy, closure of colostomy or ileostomy and the repair of the incisional hernia which almost invariably occurs after drainage cases. Today these cases should all be afforded the benefit of a trial on Wangensteen suction, for the inflammatory type of obstruction is the type that responds most readily to this method. Four of our patients died of intestinal obstruction.

HEMORRHAGE

Hemorrhage following an appendectomy is a very rare complication. We invariably invert the stump of the appendix. One must take care not to puncture the small blood vessels in the wall of the cecum. If this is done a rapidly forming hematoma may result. Several years ago I operated on a man, twenty-one years old, in whom this complication developed. At reoperation an enormous hematoma of the mesentery was found and he died despite the use of blood transfusions.

Secondary abscesses occur in neglected cases or those that have been delayed too long for operation. Secondary collections should be opened wherever they occur. The usual sites are the left iliac fossa, the subhepatic and suphrenic spaces.

MORTALITY AND STATISTICS

Out of the total number of operations performed there were seventy-one deaths (1.65 per cent). Of these there was a total of 2459 acute cases of appendicitis operated upon; thirty-three cases (1.34 per cent) of acute, perforated, gangrenous appendicitis, complicated by general peritonitis died. Thirty-four (0.79 per cent) cases died, not as a result of appendicitis but following the primary condition for which they were operated upon. Four deaths (.093 per cent) resulted from acute intestinal obstruction.

CONCLUSIONS

1. Chronic appendicitis is a distinct entity.

2. The diagnosis of appendicitis in children is often difficult to make.

3. In my experience the McBurney incision has not decreased the mortality following operations for acute appendicitis.

4. I have never regretted using the straight incision provided it is placed properly over the site of the diseased appendix.

5. While sulfa drugs have made an unusual contribution in the outcome of patients suffering from acute gangrenous, perforated appendicitis, I have not used it a sufficient number of times to present confirmatory evidence of its efficacy. The great majority of these cases recovered before the advent of these drugs in 1937.

6. With added experience less drainage of acute cases is used.

7. Excepting children spinal anesthesia is used in all acute cases.

8. Some of the complications are discussed.

9. Out of 2,459 acute cases, thirty-three patients died (1.34 per cent).

THE syndrome of chronic ulcerative colitis may develop as a result of infection by one or more of several bacterial and animal parasites, or in association with certain dietary and constitutional deficiencies, or as a result of poisoning with certain chemicals.

SULFONAMIDES IN FRESH AND CONTAMINATED WOUNDS*

MODE OF APPLICATION

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EVIDENCE is accumulating that certain conceptions in surgery, hitherto considered fundamental, must be greatly modified in the light of new experiences in the treatment of contaminated and infected wounds by the local and systemic administration of the sulfa compounds. Strode[1] following the Pearl Harbor debacle, reports the prolongation of the "golden period" for the débridement and closure of wounds from six to seventy-two hours by the immediate application of the sulfonamides to the wounded area. At the Mare Island Naval Hospital we have been able successfully to repair divided tendons twenty-six hours after the accident, administering the drugs locally at the time of repair and orally after the operation. At the same hospital, injuries to soft parts, compound fractures, compound injuries to skull and brain, have been treated, when first seen, by the local application of the sulfonamides, followed at operation by irrigation, débridement or excision, and again by the local application of the drugs, supplemented after operation by their systemic administration. Repeatedly, such treatment has resulted in the phenomenal healing of apparently hopelessly contaminated wounds. It is increasingly apparent, however, that the mode of application of these drugs is one of the most important factors in determining their effectiveness as miracle workers. Too often their administration is deferred until the patient reaches the operating table, and here, too, the mode of application may not be productive of the best possible results.

The following precautions and procedures appear fundamental at the present time, though subject to change as fresh knowledge concerning these potent drugs is obtained:

1. For local application, finely powdered or finely crystalline sulfathiazole and sulfanilamide are used in a mixture of equal parts (a) sulfanilamide, for its comparatively quick and easier solubility and therefore more prompt absorption (b) sulfathiazole, for its more prolonged action due to less solubility. Sulfanilamide and not sulfathiazole is used intracranially, due to the harmful inflammatory reaction initiated in brain tissue by the latter.

2. Whenever possible, this mixture should be applied immediately after the injury, at the first dressing by "first aider." nurse, or doctor. The fighting men themselves, stretcher bearers, corpsmen, nurses and doctors should be taught to introduce 15 Gm. of the mixture, (or of sulfanilamide, or of sulfathiazole if only one is available), directly into any large wound at the earliest opportunity—if possible the moment after it is sustained, or when first seen, or at the time of applying the first field dressing, or while awaiting transportation to an emergency station. A determined effort should be made to introduce it into the depths of the wound and under flaps of avulsed muscle or skin.

3. Immediately upon arrival at the emergency station or at the hospital, when

* The opinions contained herein are the private ones of the author, and are not to be construed as official or reflecting the views of the Navy Department, or the naval service at large.

the wound is being inspected to determine its character and extent for proper disposition of the case, the drugs should again be applied in generous amounts, directly or by atomizer, to every pocket and crevice of the wound, under elevated or avulsed flaps of skin, in exposed intermuscular planes, around denuded bone and among bone fragments. The prompt application of the drugs to *all* contaminated surfaces at the earliest possible moment, even before treatment for shock is instituted, will lead to their early absorption and effective concentration in those tissue fluids most heavily exposed to bacterial contamination, thus providing for the early inhibition of bacterial growth. In the meantime, preparations for the treatment of shock, or arrangements for the removal of the patient to the operating room, are being made.

4. At the operating table, following thorough irrigation of the wound, the local application of the drugs should not be deferred to the end of the operation, but should proceed simultaneously with the débridement or excision of the traumatized tissue. As soon as freshly incised, raw areas are exposed by the excision of tissue, the powder mixture is gently rubbed over the newly produced surfaces. Mixed with blood and tissue fluids, the powder makes a milky suspension, which is easily brought into contact with all crevices of potentially contaminated tissues. Simple "frosting" of the wound, as advocated so universally, is not equally effective in bringing the powder into contact with *all* contaminated tissues.

This periodic application of the drugs in stages, as the operation proceeds, insures their presence in the tissues almost simultaneously with, or even before, the contamination of these same tissues and tissue fluids by bacteria. It also insures that no crevice or pocket of the wound is missed. In operating in the presence of grossly infected tissues as in the repair of a colostomy or of an intestinal fistula, I have made it a rule to apply the powder every few minutes, whenever a new area of freshly incised tissue is exposed, whether it be in the abdominal wall, the mesentery, or the retroperitoneal spaces. In open resections of the bowel, or stomach, the powdered mixture is applied to all raw mesenteric areas before entering the lumen of the viscus in order that the inhibiting, antibacterial agent is present in the tissues *before* their contamination by bacteria. Similarly, in pneumonectomy and lobectomy, after mobilization of the hilum and ligation and division of the hilar vessels, the powdered suspension is brought into contact with mediastinal surfaces, the exposed pleura, and the exposed edges of the incision in the thoracic wall *before* opening the infected bronchus. In this way operative areas are impregnated with the drug one and two hours earlier than would be the case if application of the drug were deferred to the end of the operation Moreover, the impregnation of tissue fluids with the sulfa mixture before their contamination by bacteria, which may be inevitable as the operation proceeds, is, I believe, a most important factor in the inhibition of bacterial growth.

5. It is important in the final local application of the sulfonamides to avoid large dry masses and clumps of powder in the wound. To dump large amounts of the dry powder into a wound or into the peritoneal cavity, or as one author mistakenly advises, to "fill the wound with powder" is almost certainly inviting poor healing due to the presence of this dry or caked powder acting as a foreign body. An emulsion or suspension of the powdered drug made with blood and tissue lymph, thinly smeared over all surfaces of the wound, insures their early solution and effective absorption with the least interference with healing.

6. In those instances in which contamination has already been followed by well advanced infection as in peritonitis, the drugs must be applied to all infected peritoneal surfaces and pockets and such local application should be supplemented

immediately by the intravenous injection of 5 Gm. of one of the sodium salts, preferably sodium sulfathiazole. This can be done at the operating table preceding the intravenous administration of salt solution for the replacement of fluids. Following operation, if nausea or gastric suction make oral administration impossible, the intravenous injection of 5 Gm. of sodium sulfathiazole dissolved in 100 cc. of distilled water may be repeated twice daily. Subcutaneously or intravenously, sulfanilamide may be administered as a 1 per cent solution: 1.5 Gm. dissolved in 150 cc. of physiological saline, brought to a boil, then cooled to body temperature, may be administered by hypodermoclysis every six hours.

In certain instances in which vomiting prevents oral administration, sulfanilamide may be administered per rectum as a suspension in water: 4 to 6 Gm. of sulfanilamide powder are stirred thoroughly in 100 cc. of tap water and introduced into the rectum by catheter every six or eight hours for twenty-four hours, and then twice daily. A concentration of 4.6 mg. per 100 cc. of blood was achieved after three such rectal instillations.

Following operation, as soon as nausea or vomiting permit, sulfathiazole or sulfanilamide may be administered orally: 4 or 6 Gm. at the initial dose and 1 Gm. every four hours thereafter. Rarely is it necessary to continue the oral administration for more than four or five days, and it should be definitely discontinued at the end of ten days, or before if marked cyanosis, or a skin eruption resembling scarlet fever, or a high unexplained fever supervene. It is important to avoid overconcentration of the drugs in the urine with the possible production of sulfa crystals in the renal pelvis, ureter or bladder. Sufficient fluids must be administered to insure a urinary output of at least 1,000 cc. daily, although 1,500 cc. is preferable. The deposit of crystals in the kidneys will be further prevented if the hydrogen ion concentration of the urine is maintained between 7.2 and 7.4 by the simultaneous administration of 1 Gm. of sodium bicarbonate with each gram of the drug.

The following case report illustrates the use of the drugs in the management of a truly *generalized* peritonitis following the rupture of a duodenal ulcer at least twenty-seven hours and possibly thirty-six hours previously. Under ordinary circumstances such a generalized peritonitis with bilateral subphrenic collections of pus would have in all probability ended fatally:

CASE REPORT

The patient, forty-six years old, re-enlisted in the Navy on Monday, May 11, and that evening he embarked upon a prolonged celebration. Events in the next eighteen hours are remembered only hazily, but he recalls being awakened Tuesday morning with severe intermittent pains over the entire abdomen. However, he slept most of the day and when he finally awoke at 4:00, he was conscious of severe abdominal pain. He was admitted to Mare Island Naval Hospital at 11:00 P.M. Tuesday evening. There had been no vomiting or diarrhea and there had been no previous symptoms of ulcer. There was a history of syphilis in 1925, probably inadequately treated.

Physical examination disclosed a rigid abdomen, most pronounced in the epigastrium. The pulse rate was 102, temperature 101°F. blood pressure 140/90, hemoglobin 90 per cent, red blood cells numbered 4,720,000, white blood cells 21,000, 86 per cent being polymorphonuclears of which 65 per cent were mature and 21 per cent banded, blood alcohol 0.5 mg. per cent.

The admitting officer's tentative diagnosis was either a perforated ulcer or tabetic crisis. Re-examination at 6:00 A.M. Wednesday, revealed the presence of occasional abdominal cramps and severe, generalized, abdominal tenderness and rigidity with some distention. The pulse rate was 112, temperature 99.7°F. and the white cell count now numbered 27,400, 81 per cent being polymorphonuclears, of which 47 per cent were mature, 34 per cent banded. Roentgenograms disclosed a layer of air under *each* diaphragm, fully 2.5 cm. wide.

Laparotomy for a ruptured viscus was performed under gas anesthesia at 11:00 A.M. Wednesday, twenty-seven hours after the known onset of abdominal pain. The appear-

ance of the intra-abdominal viscera at operation suggested that perforation probably had occurred even hours earlier, but was not noted by the patient because of his state of inebriation. The operation was conducted through a short, high, right rectus incision. On opening the peritoneum there was an explosive discharge of gas. The peritoneal surfaces everywhere were injected and covered with fibrin. From under each diaphragm about 250 cc. of brownish turbid fluid were evacuated. On elevating the right lobe of the liver, a leaking perforation of the duodenum was found, 4 mm. in diameter, surrounded by inflamed indurated tissue and bathed in thick purulent exudate. The duodenum and surrounding tissues were rigid with induration over an area fully 10 cm. in diameter. On exploring the pelvis, a large amount of purulent material was found with heavy deposits of fibrin on the wall of the intestines lying there. To facilitate withdrawing this fluid a short muscle-splitting incision was made in the right flank, through which pus was evacuated by suction from the pelvis, both flanks, and between loops of small bowel. The perforation was closed with a flap of omentum anchored in place with mattress sutures of silk, no attempt being made to approximate the edges of the perforation itself. A mixture of 10 Gm. of sulfathiazole and 15 Gm. of sulfanilamide was applied to the inflamed peritoneum. Successive portions of this powder, placed in the palm of the operator's gloved hand were mixed with peritoneal fluid and applied successively to the peritoneal surfaces in the pelvis, both flanks, under both surfaces of the diaphragm and over the surfaces of the liver, the stomach, the spleen and the intestines. Both incisions were closed without drainage.

During the operation, the patient received 250 cc. of blood plasma, 800 cc. of normal saline solution and 4 Gm. of sodium sulfapyradine intravenously. At the end of the operation the pulse rate was 130, respirations 32, blood pressure 104/80. Following the cessation of the anesthetic, he immediately became very cyanotic with a pulse rate of 152 and respirations of 56. He was placed in an oxygen tent in which he remained for two days. Continuous suction through a Levine tube kept the stomach empty. He was permitted to drink two to three ounces of water at frequent intervals, provided it drained promptly through the Levine tube. During the first twenty-four hours, postoperatively, he drank 2,500 cc. of water, but during the same period 2,600 cc. of fluid was withdrawn from the stomach by Wangensteen suction. This concession of permitting a patient to drink water or even tea, following the suturing of a perforation, or resection of the stomach, does no harm in the presence of a well functioning suction apparatus, and it relieves the intolerable discomfort of a dry mouth. Moreover, it keeps the throat and esophagus well washed, an important precaution against ulceration of the pharynx by the indwelling tube. A negative balance, that is, less fluid withdrawn than taken in, was noted on the third day postoperatively, and the tube was withdrawn on the fourth day. Rectal instillations of 300 cc. of normal salt solution every four hours, and the twice daily intravenous administration of 1,000 cc. of 5 per cent glucose solution in normal salt solution provided an adequate fluid intake.

The accompanying chart records the amount of sulfonamides administered daily and their concentration in the blood. (Table 1.)

Table 1

Date and Hour		Sulfonamides Administered, Gm.	Blood Level of Sulfa Drugs, Mg. Per Cent
5-13-42:	1100	25 I.P. 5 I.V.	
	1500		29.5
	1700		25
5-14-42:	1200	5 I.V.	15
	1600		20
5-15-42:	0900	5 I.V.	6.2
	1630	5 I.V.	10
	2000		18
5-16-42:	0800	5 I.V.	4.5
	1600		3.9
5-17-42:	0800		1
	1430	2 (M) 5 I.V.	
	1800	2 (M)	8.3
5-18-42:	0830	2 (M), t.i.d.	6.5
5-19-42:	0900	2 (M), t.i.d.	1
5-20-42:	0800	1 (M), q.4.h.	3
5-21-42:	0830	1 (M), q.4.h.	3.5
5-22-42:	0700		2.8
Soft diet			

On the sixth postoperative day the wound partially broke open with the discharge of considerable pus. The wound healed by granu-

lation and at the present time he has a definite though moderate hernia.

The culture of the intra-abdominal pus yielded Staphylococcus aureus and a non-hemolytic streptococcus.

In the convalescent period, his only remaining nine badly infected teeth were extracted. He has had absolutely no symptoms referable to the duodenal ulceration, and he ate a soft diet compatible with the edentulous condition of his mouth. Roentgenographic studies two months after operation disclosed no gastric retention, but a persistent deformity and irregularity of the duodenum. The patient considered himself well except for the small ventral hernia, easily controlled with an abdominal binder.

SUMMARY

1. In the management of contaminated wounds and potentially contaminated wounds a mixture of equal parts of sulfanilamide and sulfathiazole powder or crystals is applied in generous amounts to every pocket and crevice of the wound at the earliest possible moment whether on the field of battle, at first aid stations, or in the emergency rooms of our hospitals. At the first dressing or at the first inspection of the wound, every effort should be made to get the drug in contact with *all* raw surfaces. The earlier the drug can be applied after bacterial contamination, the more effective will be the inhibition of bacterial growth.

2. The local application of the drug should be repeated again at operation when a débridement or excision is performed. When operating in a dirty or potentially contaminated wound as during a débridement, or in the closure of a colostomy, the drug mixture should be applied at successive stages as the operation proceeds and as freshly incised areas are exposed in the operative field, endeavoring to impregnate these raw areas simultaneously with or even before their contamination by bacterial organisms.

3. In open resections of any part of the intestinal canal, or in lobectomy or pneumonectomy, the raw surfaces of incised tissues should be impregnated with the drugs *before* opening the unsterile or infected viscus or bronchus.

4. In localized or general peritonitis, the drug should be brought into contact with all contaminated surfaces. Mixed with blood and tissue fluids, the drug is thinly smeared or rubbed over all infected peritoneal surfaces, thus insuring maximum absorption and least interference with healing. Dumping large masses of the dry powder into a wound is inviting poor healing, as it may then act as a foreign body.

5. "Frosting" a wound reaches only the superficial surfaces of a wound. A suspension of the powder in tissue fluids permits more effective contact with all nooks and crannies, all crevices of a wound.

6. After operation, when vomiting or gastric suction prevent their oral administration, the drugs may be administered subcutaneously, intravenously, or rectally:

As a 0.8 per cent solution (8 Gm. in 1,000 cc. Ringer's or normal saline solution) sulfanilamide may be administered by hypodermoclysis.

Five Gm. of sodium sulfathiazole dissolved in 100 cc. of distilled water may be given intravenously twice daily. Sulfanilamide may be given intravenously every six hours as a 1 per cent solution (1.5 Gm. in 150 cc. normal saline solution).

Four to 6 Gm. of sulfanilamide powder suspended in 100 cc. of tap water may be administered per rectum as a retention enema every six to eight hours for twenty-four hours, and then twice daily.

7. Orally, 4 to 6 Gm. of the sulfonamides may be given as the initial dose, and 1 Gm. every four hours thereafter. Rarely should it be continued in this dosage more than five days, and very rarely after ten days. Marked cyanosis, a scarletiniform rash, or a high unexplained fever demand the discontinuance of the drug. A daily urinary output of at least 1,000 cc. is imperative and 1,500 cc. is preferable.

8. Many cases including compound injuries of the extremities, skull, thorax and

abdomen, have been treated successfully according to the above principles. The complete recovery of a case of generalized peritonitis with large collections of pus in the pelvis, in both flanks, and under both leaves of the diaphragm, following the rupture of a duodenal ulcer twenty-seven hours previous to operation, is presented in detail. In this instance, the concentration of the sulfonamides in the blood reached 29 mg. per cent in the course of the first twenty-four hours after operation.

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TREATMENT OF SKELETAL PAIN WITH PROCAINE INJECTIONS

AN ANALYSIS OF 295 CASES IN GENERAL PRACTICE

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IN the course of an average general practice, 295 patients were injected with procaine, nupercaine or eucupin solution. The magnitude of the problem of pain can be realized when one finds that 214 additional patients complaining of pain during the same five-year period were not injected, i.e., the former group had pain severe enough so that an injection, often feared and usually detested by the layman, was not objected to.

Causes of Pain. Table 1 lists the causes of pain as determined by myself and various consultants, and proved by (1) operation in a few cases, (2) roentgenologic study, (3) physical examination and (4) passage of time, which indicated that a serious underlying condition was not present. Consultation with orthopedists, neurosurgeons, internists and general surgeons at the Mayo Clinic and elsewhere was obtained.

TABLE 1

I. Trauma	
A. Direct injury and bruising	13
B. Indirect twisting and straining	
1. Sprained ankle	24
2. Sprained back	6
3. Sprained knee	2
II. Protruded intervertebral disc	
A. Suspected in 9, removed in	3
III. "Stiff neck"	17
IV. Painful scar	6
V. Osteo-arthritis	31
VI. Rheumatic (myalgia, fibrositis, neuralgia)	164
A. Predominantly menopausal	9
VII. Miscellaneous: Recurrent painful ankle	2
Herpes zoster, persistent	1
Pleurisy, radiologically proved	4
Lung abscess	2
One each of gout, renal colic, gallbladder colic, carcinoma of stomach	4

Each figure represents one patient, classified under the predominant cause of pain.

One must remember that a patient with proved hypertrophic arthritis of the spine does not have pain because of the bony changes themselves, inasmuch as similar roentgen views can be found in patients who have no pain. The same statement holds for patients with bony spurs of the heel.

Because of lack of definite diagnostic criteria for such diagnoses, the terms lumbago, fibrositis, myalgia, lumbosacral strain and the various neuralgias have not been used.

PAIN IS NOT DIAGNOSTIC

The more one injects patients complaining of pain, the more one realizes that certain types of pain are not diagnostic and far from being pathognomonic. Precordial pain radiated to the left shoulder and down the left arm is an especially common cause of worry to the patient because of its possible association with angina pectoris. Chest pain, of "pleuritic" type, i.e., increased by deep respiration or cough, is quite often of superficial origin, as indicated by the lack of findings on physical and roentgenologic examination.

Pain in the low back is a common complaint. After elimination of pelvic, bladder, rectal, kidney and vertebral causes, there remains a large number of individuals who complain of low back pain in acute attacks following slight or heavy exertion or in a chronic form. The great majority of these individuals can be relieved by local anesthetic injections, exercises, heat and reduction in weight. Belts were not recommended for any patient with back

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pain, except for such times (riding a tractor or a railroad engine) when persistent jarring and strain could be expected. A belt or corset is no more a treatment for back pain than a crutch is a treatment for a painful ankle.

The relief of pain by procaine injections is not diagnostic. The pain of a protruded intervertebral disc can be relieved for weeks. A woman with a giant cell tumor of the knee and painful disability on walking was relieved by such injections. A surgically proved case of spasm of the sphincter of Vater was relieved more quickly by infiltration of the tender point over the right upper quadrant than by nitroglycerine and morphine. Abdominal pain due to a cystoscopically proved enlargement of the verumontanum and urinary retention was relieved promptly by injection. *The relief of pain by local anesthetic injections does not free one from the responsibility of determining a possible serious cause for the pain.*

Repeated recurrences of pain indicate that (1) the underlying condition has not been found, (2) auxiliary treatment has not been carried out, e.g., exercises for painful backs or (3) the "trigger points" were not injected.

PHYSIOLOGIC BACKGROUND

Sir Thomas Lewis' clinical and experimental work on pain[1,2] and Leriche's[3] surgical attacks on pain-carrying nerves and the sympathetic nervous system, have founded new ideas.

Pain can be studied scientifically, because it now can be produced under standardized conditions. Lewis found that pain results from the injection of hypertonic saline solution into various structures. By injection of skin and muscle, he was able to produce different types of pain.

Skin Pain. The pricking of a needle into the skin results in a sharp, localized "sticking" pain; the same type of pain appears if saline solution is injected rapidly or when the skin is touched with a hot wire. "Burning" pain results when a prolonged stimulus (freezing or heat) is applied to the skin.

Abrasions, fine multiple scratches, freezing, ultraviolet irradiation and continued friction result in the same type of pain. The subject cannot distinguish between them but complains merely of "burning." (What does the patient mean when he complains of a burning pain?)

"Dull" pain arises from deeper structures. If hypertonic saline solution is injected into muscle, a dull, poorly localized, "aching" or "sickening" pain results, resembling that found in rheumatic and arthritic conditions, and muscle and ligament pains. Even procaine solution, if injected rapidly into muscle or fascia, will procure this dull, "full" type of distress.

Nerve Pain. A sharp, shooting pain indicates that a nerve has been involved, or that a sensitive structure such as a sheet of fascia has been touched with the needle.

TECHNIC OF INJECTION

The "trigger point" must first be found. This point can be found by palpating, in the painful area itself, around it or between it and the spinal column, for an area which is markedly tender. If the patient does not *wince* when pressure is made on a point, it is probably not the one sought. If finger tip pressure causes the patient to say, "*That* is my pain," one may confidently predict relief.

If several areas of tenderness are found, mark each with a skin marking pencil or a drop of colored antiseptic solution, then compare to determine which are the most tender and inject only those causing wincing tenderness.

Multiple areas can be treated only by multiple injections, with definitely poorer results than injection of one or two points, by infiltration of the entire area with ½ per cent procaine solution or by infrared radiation or short wave therapy.

Depth of Injection. A wheal is raised with a fine needle by injecting four or five drops directly into the skin. A No. 24 gauge

two or three inch needle is then attached, and gently inserted through the wheal and into the deeper structures until underlying bone is reached or the patient complains of pain. Two cc. of procaine is injected when pain is elicited. After a wait of one-half minute, palpation is then carried out to see if tenderness has disappeared. If no pain is produced by pressure, that "trigger" point has been treated sufficiently. If some tenderness persists, the needle is introduced farther and the process repeated. It is of clinical research interest to note at what level, and approximately in what structure, the painful area is located.

Types of Solution. Procaine solution of 1 per cent strength was used in the great majority of these injections. Nupercaine solution (1:1000) is a longer acting local anesthetic which gives better results in chronic painful conditions.

Patient's Position. Except for the most trifling injection, the patient should be kept lying down, and preferably so placed that he cannot see the needle or the injection. He should be told of the initial prick of the needle but should not be asked repeatedly if pain is present.

Nervous or apprehensive patients had best be given a small dose of barbiturate, e.g., 1½ gr. of nembutal, preceding the injection. One patient out of three will feel dizzy or faint if allowed to sit up promptly after the injection of 10 cc. or more of procaine or nupercaine solution, so that it is best to have all patients remain lying down for ten minutes thereafter.

Has Enough Solution Been Injected? The patient should assume the position or go through the motions which caused him pain previously, before one should assume that all trigger points are treated.

How Many Injections Are Needed? One injection relieves 25 per cent of muscle and ligament pains; two injections bring relief to half of the patients. The injections are given at two or three day intervals. If longer and longer periods of relief are not obtained by each injection, in the remaining group, the patient should be restudied for other causes of pain or questioned about work which may be aggravating the condition.

What Should the Patient Be Told? The patient should be informed that in most cases there is a reaction from two to six hours after the injection. At this time, the pain may recur and may temporarily be worse than previously (he should have analgesic tablets to take during this time). Following this episode, either the pain will disappear leaving only a residual stiffness or it will persist. He should be told to carry out his usual daily routine, with avoidance of heavy, straining positions or movements.

ANATOMIC BACKGROUND

Through the courtesy of Dr. T. T. Job, Professor of Anatomy at Loyola University School of Medicine, several weeks were spent in dissecting various painful areas and attempting to determine the source of pain and the structures which the injecting needle touched or penetrated. This material will be reported separately. Dr. Job's caution should be remembered: "Don't speak of muscle pain. All muscle fibers are encapsulated in fascia. There are no nerve endings in muscle except motor end plates; pain nerves are in the fascia."

SUMMARY

Procaine injections will cure the great majority of muscle, fascia and ligament pains. Relief is only temporary, though often gratifying, if an organic cause is still at work or if improper work or posture is persisted in. Such injections are of little value if trigger points are not found and each injected.

In the long term view, such injections must be considered as only a part of the treatment of osteo-arthritis and rheumatic conditions. The correction of posture, removal of foci of infection (occasionally), reduction of weight, avoidance of chilling and overwork, the daily use of "limbering up" exercises, all must be considered.

VENOGRAPHY AS AN ESSENTIAL AID IN THE TREATMENT OF VARICOSE VEINS*

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FOLLOWING the work on venography reported by Bauer,[1,2,3] Starr, Frank and Fine,[4] Fine and Sears,[5] Homans and Dougherty,[6] we adopted the procedure to study chronic conditions of the lower extremities resulting from disorders of the venous circulation. This was prompted by the occurrence of postoperative complications.[7] Treatment of these complications was costly and caused much loss of time.

Since the adoption of this procedure as an essential routine in working up cases, there has been no occasion of postoperative complication in our last series of 148 cases. We found the percentage of cases rejected for treatment after venographic study was the same as the percentage of postoperative complications reported in a previous publication.[8]

Information gained by our study afforded us increased knowledge of the anatomy and physiology of the venous circulation.[9] One fact observed by fluorscopic examination during injection of the diadrast with the patient in the supine position, was the rapidity of the dye seeking the deeper avenues of return as early as was possible, regardless of which distal small superficial vein on the dorsum of the foot was selected as the site for injection. This fact obviates the use of the tourniquet technic as described by Mahorner.[10] We believe by the use of the tourniquet any superficial phlebitis of the lowermost part of the leg and foot is not revealed. We do believe with him that it is of advantage in trying to ascertain distal deep phlebitis in cases postoperatively and in those in which there exists an old ulcer with a thrombophlebitic history.

Another fact learned was that one could not place utmost reliance on the value of the generally accepted tests used to determine the status of the deep venous circulation. This applies to the tests we routinely employ, namely, Trendelenburg, Perthes, Oscher-Mahorner, Pratt and Heyerdale, recently reported.[11] In three instances complications occurred when Veal's venous pressure studies were made.

The fallacy encountered in these tests is the inability to establish the existence of a very small segment of the deep venous system involved by an old thrombotic process which lights up after massive injection. Likewise we have seen many instances of retrograde flow occur in veins with good functioning valves and no evident disorder to explain the change of flow. This finding has never been proved to produce ill effects after injection.

X-RAY TECHNIC

The dye used in diodrast 35 per cent. The Dolan mouth test prior to injection is given the patient. Twenty cc. of the dye is given slowly through a small needle (No. 26 to 27 gauge) over a period of approximately two minutes into a small vein on the distal portion of the dorsum of the foot below the external or internal malleolus.

In our own experience, it has only occasionally been necessary to cut down on a vein to inject the dye. Such cases have had marked edema or swelling of foot and ankle, and it was not possible to find a vein without incision. All patients are placed in the supine position. Changing

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angles of the table, Trendelenburg and opposite, have proved to be disadvantageous in visualizing upper thigh and iliac veins.

Two lead screens placed on either side of the top of the Bucky table have been used recently. These are of sufficient width and are so placed that the intervening space allows the coverage of half a 14 by 17 film. These lead screens are long enough to reach from the ankle to the lower abdomen. The leg can be satisfactorily positioned between these screens so that the entire venous circulation of the leg, thigh and lower pelvis that we wish to visualize can be portrayed on serial films.

During the time of the two minutes' injection, six exposures are made. Two exposures of the leg are made from approximately the ankle upward after about 4 to 5 cc. of the dye has been injected. As soon as the tube stand and Bucky tray can be shifted upward, two more exposures are made of the upper leg, knee, and lower thigh. Again, the tube stand and Bucky table are shifted upward and two more exposures are made which include the upper thigh and lower pelvis. These exposures are in pairs and each exposure is made from a stereoscopic position.

We realize that stereoscopic examination of any part requires that that part be absolutely still for a proper stereoscopic view. However, the dye remains long enough in one place to allow us to differentiate between the deep and superficial circulation. This has proved to be very important and is very difficult to demonstrate by single film examination. By rapid work, it is possible to complete six of these exposures in the time that the dye is injected. Occasionally, a fourth film is made with two exposures, one on either half of the film, after the conclusion of the injection. Usually, this film is made of the upper leg and knee area.

By such procedure, we obtain a series of overlapping films of the region from the ankle up to and including the lower pelvis. The time interval between these films is such that in the normal case, visualization of the venous structure of leg and thigh into which the dye enters is obtained. The fourth film gives us added information about the upward passage of the dye in the upper portion of the leg. The stereoscopic film enables us with considerable certainty to reconstruct anatomical positions.

SUMMARY

We believe the use of serial venography affords valuable information in the study of the deep venous circulation of the lower extremities. Since this procedure is simple, free from contraindication to its use in the face of any disorder, and because of the great benefit we have derived in avoiding postoperative complications, we do not hesitate to state that no preoperative study of the deep venous circulation is complete without the use of venography.

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Case Reports

ABDOMINAL PREGNANCY—FIFTH MONTH*

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BEFORE the days of careful histological study of specimens removed at operation, it was believed that the ovum could be implanted upon any portion of the peritoneum with a resultant primary abdominal pregnancy. With careful study of the material, it is now conceded that most abdominal pregnancies are the result of a ruptured tubal pregnancy and are, therefore, secondary rather than primary abdominal pregnancies. The hemorrhagic complications of this condition may be extremely severe as exemplified by the case reported.

CASE REPORT

Mrs. M. R., a thirty-eight year old married, white woman, was brought to the Malden Hospital as an emergency patient, following a vaginal examination in a physician's office on April 17, 1942. She was in shock and complained of severe right lower quadrant pain associated with nausea and vomiting. She had been perfectly well up to three months prior to admission to the hospital, when she noticed a constant dull aching sensation in the right lower quadrant. The pain was not relieved by rest, it was not increased by exercise, nor did it radiate in any direction. Two months before admission she had an attack of sharp severe pain which started in her right back and which radiated forward to the right lower quadrant of the abdomen. This lasted ten minutes and disappeared after rest in bed. At approximately the same time she noticed a constant vaginal discharge, which had not been observed previously. Over a period of four weeks, the attacks increased in number, severity and duration, and during one very severe attack, there was swelling of the abdomen, this lasting from twelve to fourteen hours. Six weeks before admission, there was observed a small lump in the right lower quadrant which gradually increased in size. She consulted her physician and was told that she was either pregnant or that she had a tumor. He treated her by means of injections which did not relieve her symptoms. One month before admission she had a sudden vaginal hemorrhage, passed approximately a cupful of blood, and was subsequently aware of a daily, dark brown stain on her underclothing. For the last month the pain in the right lower quadrant constantly increased in severity and became so frequent that it was necessary for her to spend most of her time in a reclining position in bed or on a couch. Between the attacks of severe pain she always experienced a dull ache. During these four weeks she noticed that her lower abdomen had increased approximately twice its normal size, and the mass was directed downward and toward the median line. On the day of admission to the hospital she consulted another physician who told her that she was seven months' pregnant. While seated in the doctor's office she felt suddenly faint and collapsed. Upon recovery from her syncope she was seized with terrific right lower quadrant pain, which radiated to the back, this pain being more severe than any she had previously experienced. She also felt nauseated and vomited two or three times, she had a marked sense of weakness and prostration, with difficulty in breathing, and she had a sense of impending death.

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Her periods had always been quite regular, lasting seven to ten days, and were accompanied with marked dysmenorrhea in the form of right lower quadrant pain.

Fig. 1. Fetus, well developed and well nourished, weighing 530 Gm. Several cm. of cord are still attached.

The patient, whose husband is living and well, had had two previous pregnancies; the first child was seventeen, and the second fifteen years of age. Her mother had died from a disease unknown to her, and her father from influenza. One sister had died in childbirth, and four brothers and three sisters were living and well. There had been no familial diseases. She had had the usual diseases of childhood, and rheumatic fever at the age of fifteen years; this necessitated her remaining in bed three months, and she was told that she had a heart involvement. Subsequently she was examined by five physicians who informed her that her heart showed no abnormalities, and there had been no further cardiac symptoms. There had been infrequent attacks of tonsillitis. Fifteen months before admission to the hospital she had an attack of polyarthritis, accompanied by swelling, pain and redness, and involving both elbows and both knees. Bed rest for approximately two and one-half months arrested the condition, and there had been no further attacks. There had been no other hospital admissions or operations.

The patient was seen as a thirty-eight year old woman lying in bed in apparent great pain; she was pale and dyspneic; there were beads of perspiration on her forehead; her extremities were cold and clammy and she was apparently in profound shock. The head was grossly normal; the chest was symmetrical; the lungs were resonant and clear throughout; the heart showed the typical signs of mitral stenosis; the pulse was so rapid it could not be counted, and the blood pressure was unobtainable. The abdomen showed a large mass which rose above the umbilicus, occupying the right lower quadrant and part of the left; it was extremely tender to palpation and there was definite spasm of the muscles over that area. There

Fig. 2. Fetus with attached cord and placenta. The cord is of average diameter, but somewhat shorter than normal. The marginal attachment of the cord is clearly shown. Along this margin, the chorionic villi form a thick fringe and project freely beyond the chorion and amnion.

FIG. 3. Placenta, weighing 275 Gm. This picture shows the maternal surface with a short segment of cord emerging from one side. Along one margin, the chorionic villi project in coarse masses. The villi are delicate and show no tendency toward cyst formation. The remaining maternal surface is comparatively smooth, but coarse and leathery.

FIG. 4. This resembles Figure 3, but in addition, shows the relative position of the buried tube within the substance of the placental mass. This is indicated by a sound which has been passed through the tube to emerge at both the proximal and the distal ends of the tube. The tube was deeply hidden and was only uncovered after making linear parallel incisions into the thicker portion of the placenta.

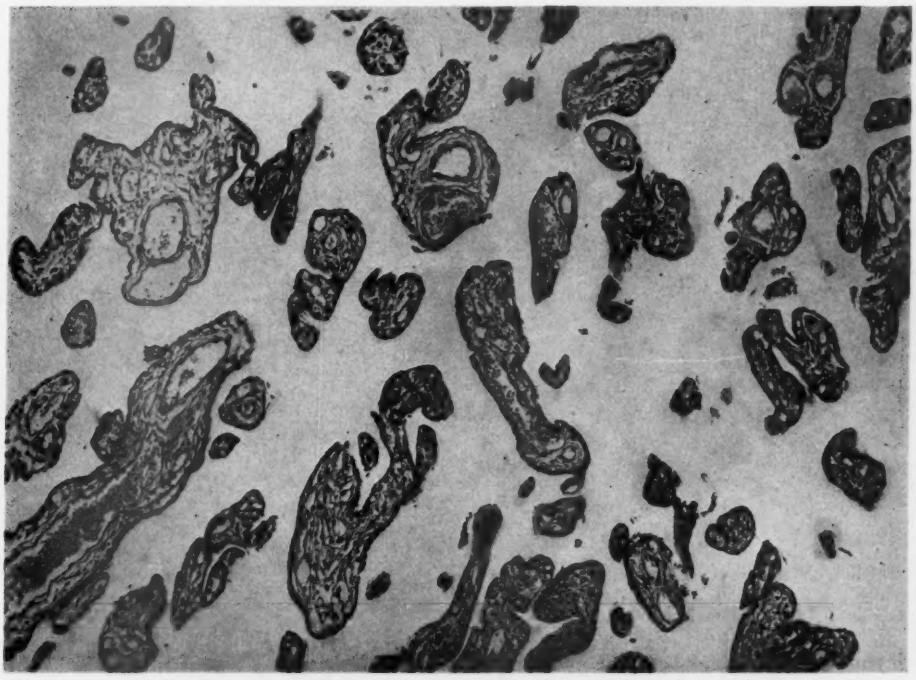

FIG. 5. Many healthy chorionic villi seen in longitudinal and cross-section. Such tissue is representative of the mass of villi projecting from the chorion adjacent to the attachment of the cord. Each villus is well vascularized and in many, the vessels are overdistended. The stroma is loose but not edematous. The inner layer of trophoblastic cells—Langhans cells, has disappeared, and the outer syncytial cells are flat and inconspicuous. The intervillous spaces are patent and free of fibrin. × 108.

was no tenderness in the upper part of the abdomen and peristalsis was not observed at the time of examination. A vaginal examination was not done because of a steady trickle of blood through the vagina. Her red blood count was 3,000,000, and her hemoglobin, (Sahli) 40 per cent.

After her admission to the hospital she was seen by several members of the visiting staff who agreed that she had had a lower abdominal accident with hemorrhage, and that the abdomen should be explored after her shock had been combatted.

She was placed in the Trendelenburg position; heaters and blankets were applied and morphine sulfate, gr. 1/6 (10 mg.) was administered. She was given 500 cc. of blood plasma, 1000 cc. of normal saline solution and 500 cc. of citrated blood intravenously. With this treatment she gradually came out of her shock and she continued to improve, although her pulse remained rapid and thready. Fifteen hours after the admission the blood pressure was 120 systolic and 70 diastolic and the pulse, although elevated in rate, was strong in character. During her stay in the hospital the abdomen became progressively larger and duller, there was greater tenderness and the spasticity involved the entire abdominal wall. At this time one of us, (L. E. P.), was called in consultation. The patient was then pale but not in shock; the pulse was weak in character and 140 in rate; and the abdomen was distended and boardlike, making it impossible to feel any individual structures through the abdominal wall. A vaginal examination re-

FIG. 6. Section through an area of the placental mass adjacent to the tube. This picture clearly shows ovarian tissue in the upper two-thirds of the block, and fibrin, disintegrating red blood cells, and the shadowy outlines of necrotic villi in the lower third. There is very little attempt at organization of this mass of blood and fibrin. A dense band of fibrous tissue separates the placental tissue from the ovary. Above this, the stroma of the ovary is edematous. A medium-sized vein, moderately dilated, lies just above the center of the field, and just above and immediately adjacent to the latter, lies a hyalinized corpus luteum. There is no decidual reaction within or along the surface of the ovary. × 24.

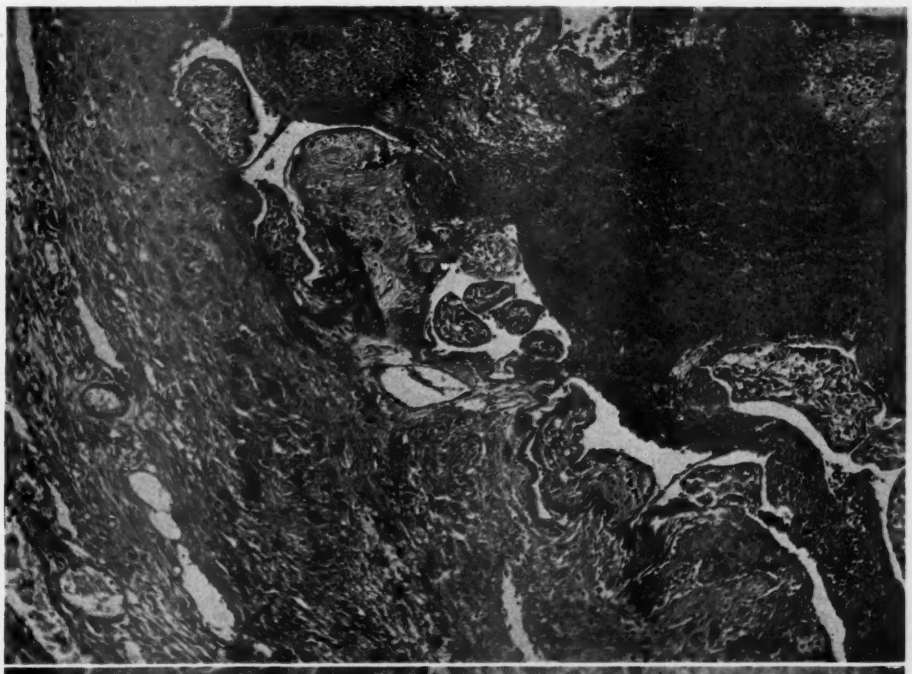

Fig. 7.

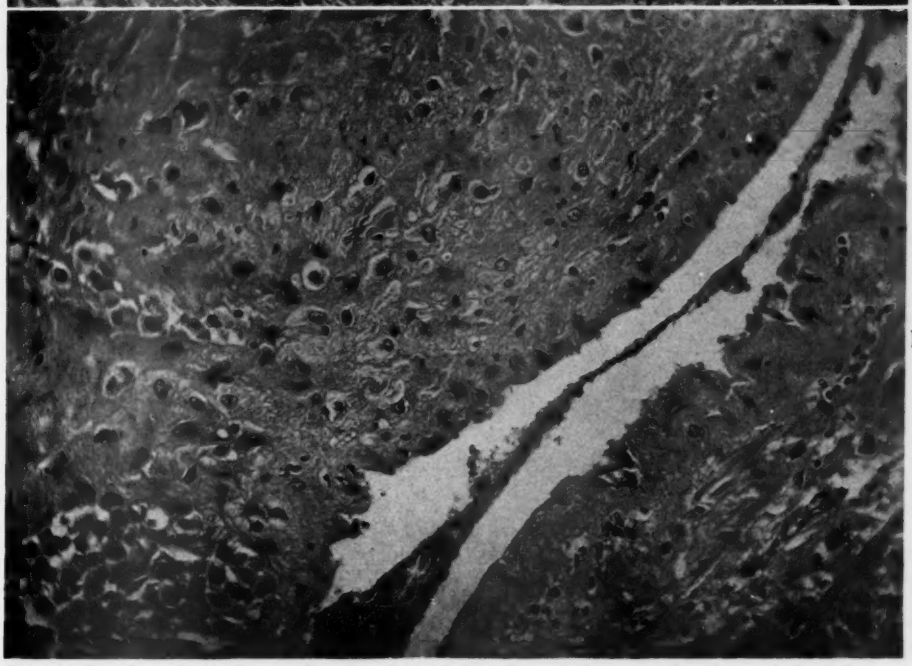

Fig. 8.

Fig. 7. Section through another portion of the placental mass, showing on the left one surface of broad ligament and on the right, chorionic villi, intervillous spaces, and blood clot. This area of broad ligament is made up of edematous fibrous tissue, scattered vessels and a moderately well demarcated zone of decidual cells. No serosal cells are visible, but a delicate layer of dense fibrin resembling a Nitabusch membrane tends to separate these decidua cells from the adjacent villi. Here the villi are collapsed, but they are still vascularized and apparently capable of functioning. Much of the intervillous space is filled with a moderately fresh coagulum. × 108.

Fig. 8. Another section through the junction of broad ligament and placenta. A moderately dilated blood sinus divides the section diagonally and unevenly. This is lined with endothelium and bordered above, and on the left side, by decidua and below and on the right, by edematous ground substance and hypertrophied smooth muscle fibers infiltrated with lymphocytes and histiocytes. A small tongue-like mass of trophoblasts project into the lumen and another nest of trophoblastic cells is embedded among decidua cells in the lower left-hand corner of the section. × 220.

vealed moderate vaginal bleeding and bulging and tenderness in the cul-de-sac of Douglas. A preoperative diagnosis of abdominal pregnancy had been made by Dr. T. G. Tighe, the resident surgeon. At the time of examination by the consultant it was evident that there was a severe intra-abdominal hemorrhage, and the preoperative diagnosis of abdominal pregnancy was allowed to stand. The improvement in her general condition having reached its optimum, operation was decided upon and the patient was taken to the operating room.

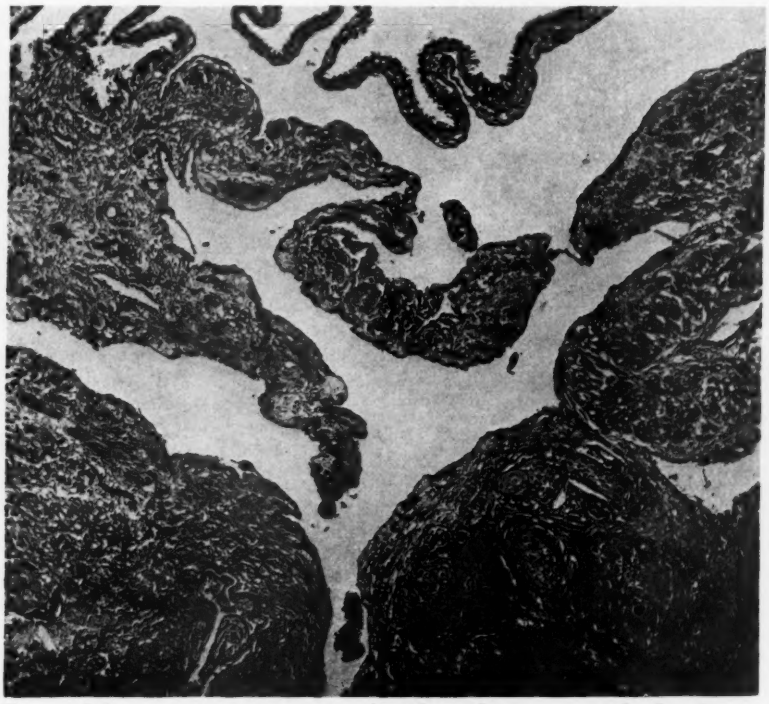

FIG. 9. A low power transverse section through a segment of tube. A narrow membrane of mucosa lies at the top of the field. This is partially separated from the underlying muscularis by large, communicating thin-walled sinuses. A mass of hypertrophied, smooth muscle fibers on the right and left border this sinus as it passes through the thickness of the tube. Below, two large clumps of fibrin, in which sclerosed chorionic villi are embedded, are almost inseparably attached to the tube. In this area, the wall of the tube is very thin and only the mucosa and blood sinuses separate the lumen of the tube from the peripherally attached placenta. × 96.

Spinal anesthesia was administered by Dr. Daniel Johnson, the hospital anesthetist. The urinary bladder was catheterized and the catheter was left *in situ*. The abdomen was prepared and the patient was placed in the Trendelenburg position. A median pelvic incision was made from the symphysis to the umbilicus. On entering the peritoneal cavity there was the escape of considerable blood, estimated at 500 cc. The pelvis and the lateral gutters were filled with free and clotted blood. The uterus was located. It was about twice its normal size; the left tube and ovary, were not enlarged, were adherent in the cul-de-sac of Douglas. On the right side there was a large mass which extended from that side of the uterus to the pelvic brim and rose above it. This mass contained an abdominal pregnancy, it was very tense, and, involved in this mass were the right side of the uterus, the right tube, the right ovary, the cecum, the appendix and the right broad ligament. In attempting to free the mass it was punctured, with the escape of amniotic fluid. A well formed, five months' female fetus, alive at the time of delivery, was delivered through the incision, and the cord was cut. The mass was shelled out from the posterior surface of the right broad ligament with the hand, the cecum and appendix were separated from it, but the right tube and ovary remained attached to it. The right infundibulo pelvic ligament was cut be-

tween clamps, the right broad ligament was clamped and cut under the mass until the right side of the uterus was reached, the tube and ovarian ligament were clamped close to the uterus and cut, and the mass was removed *in toto*. In addition to the tube and ovary the specimen contained a well formed placenta. All clamps were replaced with suture ligatures of No. 1 chromic catgut. Adhesions about the cecum and the appendix which had been severed were ligated with the same material. An area in the uterine fundus which had been bared during the dissection was covered over with serosa by means of a continuous suture of No. 1 chromic catgut. The hemostasis being complete, the blood clots in the pelvis and lateral gutters were removed but the liquid blood was left in the pelvis. The uterus was in forward position, the left adnexa, which had been freed from the cul-de-sac of Douglas, were left *in situ*, the omentum was spread over the intestines, which were distended, placed behind the uterus, and the incision was closed in two layers with a running suture of No. 1 chromic catgut in the peritoneum, and with through-and-through black silk sutures in the remaining layers. At the completion of operation the condition of the patient was no more serious than it was at the start. Upon her return to bed she was given 500 cc. of blood plasma and 500 cc. of citrated blood by transfusion.

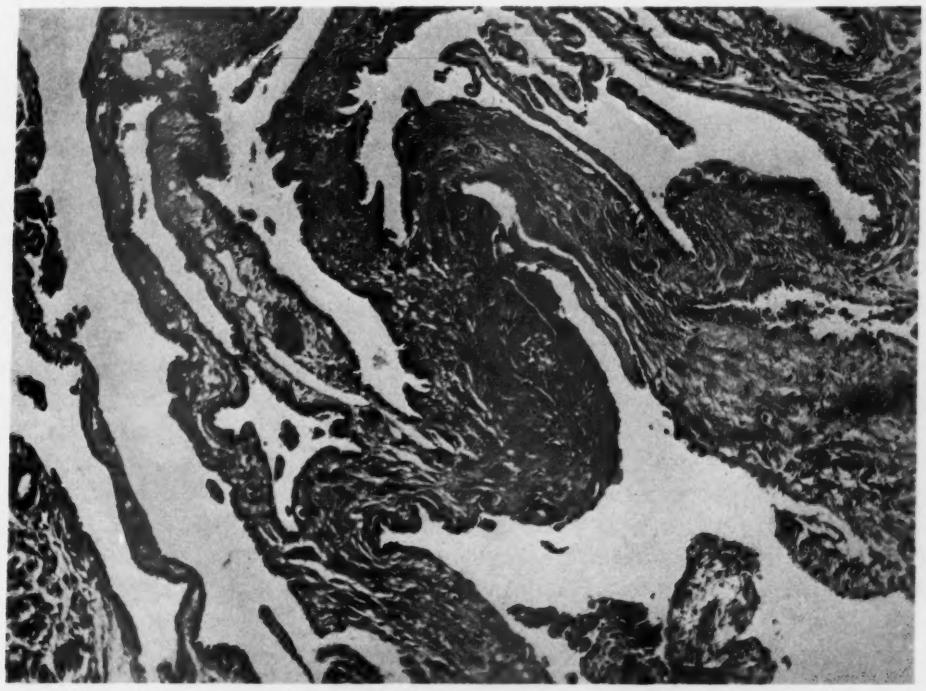

Fig. 10. A higher power magnification of a small segment of tube, showing the lumen divided up into numerous epithelial-lined spaces—a picture quite consistent with old, healed inflammatory reaction within the tube. In one area, just beneath the epithelium, there is a narrow zone of decidua cells and attached to this is an amorphous mass of fibrin. Large syncytial giant cells are embedded in the latter. This fibrin forms part of the boundary of a large intervillous space. In the lower right-hand corner of the section, several sclerosed chorionic villi project into this space. At this particular point in the tube, a layer of epithelium bordered externally by a narrow zone of decidua forms the only barrier between the lumen of the tube and the attached placenta. The smooth muscle in this area of the tube has entirely disappeared. × 108.

The convalescence was under the immediate supervision of Doctors S. P. Norman, and T. G. Tighe, Dr. Phaneuf seeing the patient only twice. Upon her return to bed, her temperature was 97°F., her pulse 150 and her respirations 34. On the first postoperative day the temperature rose to 101°F., the highest during her convalescence, the pulse was 130 and the respirations 40. At the sixth day all three had dropped to normal limits. She was allowed out of bed on the tenth day and was discharged on the thirteenth day, at which time the incision was well healed. She has remained well up to the present time.

The specimen was taken to Tufts College Medical School and submitted to Dr. H. Edward MacMahon, the Professor of Pathology, for examination. The report is as follows:

The specimens consisted of a female infant and placenta. The clinical diagnosis was abdominal pregnancy, fifth month.

Specimen No. 1. This was a well developed and well nourished white female infant, weighing 530 Gm., and measuring from head to heel 27.5 cm. Nails were formed on both hands and feet. The anus was patent. The eyelashes and eyelids were regularly formed. The head was round and symmetrical. The bones, the sutures of the calvarium, and the fontanels bore a normal relationship to each other. The ears were fully developed, well formed, and lay flat against the head. The chest, abdomen, external genitalia, and extremities showed no evidence of disease. A segment of cord, 10 cm. in length, and 1.5 cm. in width, was normally attached at the umbilicus. *Gross Diagnosis:* A healthy female infant, approximately 5½ months old.

Specimen No. 2. This was a placenta, together with 15 cm. of cord, weighing 275 Gm. The cord was regular in size, shape, color and consistency, and the cut surface presented the openings of three vessels, from which little fluid blood could be expressed. The fetal surface of the placenta was smooth, glistening, gray, and transparent, and was represented by the amnion, which could be easily detached from the underlying chorion. The placenta was nearly circular in outline and measured 20 cm. in diameter. For the most part it was thin and less than 1.0 cm. in thickness, but along one margin, it was coarse and thick, and measured 4 cm. in width. From this thick zone, the width rapidly tapered off as one passed from one side to the other, and where it was thinnest, it measured only 0.2 cm. The cord showed a solid marginal attachment, and joined the placenta at its thickest point. From here the vessels spread out in an irregular, fan-like manner. The vessels were small and inconspicuous and many were pale, collapsed, white, and empty. The maternal surface was very unusual. A small crescentric zone of grayish-red, friable, healthy chorionic villi, measuring about 2 to 3 cm. across, and 2 to 3 cm. thick, lay along one margin. This corresponded to the thickest portion of the placenta and also to the site of attachment of the cord. The remainder of the maternal surface, measuring about 18 cm. in diameter, was represented by a comparatively smooth, but cobbled, gray to reddish-gray, firm, leathery, compact, membrane-like structure, that was inseparably attached to both chorion and villi. A very careful examination of this membraneous, maternal surface, failed to reveal any recognizable structure, with the exception of a cluster of large blood vessels with thick walls and gaping lumina. These represented the distended and tortuous vessels of the broad ligament. Multiple sections through this area, which bordered the thickest portion of the placenta, revealed many more similar vessels, much edematous gray fibrous tissue, and an intact, but flattened ovary, and a completely hidden tube. The latter was tortuous, thick-walled, and 7 cm. in length. Both the inner and outer ends of the tube were discernible, and a 1 mm. probe could be easily passed through the outer two-thirds. The distal or outer end of the tube was represented by a gaping lumen that lay just at the junction between the friable and compact portion of the placenta.

Multiple sections into the ovary revealed a variable amount of edematous, gray, leathery, fibrous tissue, many vessels and several small cysts 0.3 to 0.5 cm. in diameter. There was no recent corpus luteum. On carrying these incisions more deeply through this gray, leathery, edematous, and vascular fibrous tissue of the broad ligament and ovary into the substance of the chorion itself, a narrow zone of villi and blood, representing the intervillous space, was recognizable in the thicker areas, adjacent to the attachment of the cord. The villi became less and less conspicuous, as one traveled farther from the cord, and in the thinnest portion of the placenta, they disappeared entirely. Here, only a narrow streak of coagulated blood separated the thin, flat, membraneous chorion, from an equally thin, fibrous, maternal wall. It was obvious that less than one-third of the entire maternal surface of the placenta was composed of functioning chorionic tissue. This corresponded to the thickest portion of the placenta and was confined to that area radiating out from the attachment of the cord. This area came in contact with the upper portion of the broad ligament, the vessels of the broad ligament, the tube and the ovary. *Gross Diagnosis:* Ectopic

abdominal pregnancy, with placental site covering the broad ligament, ovary, tube, and pelvic peritoneum; old, healed salpingitis; follicular cysts of the ovary; hemorrhage into the decidua; widespread infarction of the placenta.

Microscopic examination was made of eleven sections taken from the cord, placenta and placental attachments.

Section No. 1. This comprised a portion of ovary, broad ligament, and placenta. The ovary was very edematous, and the stroma showed a number of old corpora albicantes. Along one surface, there was a layer of old, coagulated blood, in which the shadowy outlines of necrotic chorionic villi were still recognizable. There were many histiocytes filled with hemosiderin in this coagulum. The blood in the form of dense, closely packed fibrin, and fragmented and crenated red blood cells, came into immediate contact with those fibroblasts which formed the cortex of the ovary. These fibroblasts were abnormally large and were embedded in fluid, rich in old blood cells and fibrin. Practically all of these fibroblasts ran parallel to the surface of the ovary and there was little or no tendency for the fibroblasts or adjacent capillaries to extend into the adherent coagulum. There was a moderate infiltration of monocytes and lymphocytes into the underlying ovarian tissue. There were no viable, trophoblastic or syncytial cells and there was no suggestion of any ingrowth of villi into the substance of the ovary. In this area, there were no recognizable decidua cells. *Diagnosis:* Ovary and adjacent broad ligament, bordered by placental tissue showed so-called placental infarction.

Section No. 2. This consisted of ovary, chorionic villi, intervillous spaces and chorion. The ovary was edematous. Its arteries and veins were dilated and thick-walled. A dense layer of fibrin was adherent along one surface and embedded in this were chorionic villi. Some of these were necrotic, others were sclerosed. They contained few vessels and these were collapsed and empty. In areas, the intervillous spaces were filled with fibrin; in other areas, they were free. The *syncytial* cells were poorly preserved. There were scattered cells in the layer of fibrin adjacent to the chorion that resembled decidua cells. The chorion itself was sclerosed, and its vessels were partially occluded. The intervillous space was unusually narrow. *Diagnosis:* Ovary, bordered by an area of completely and partially infarcted placental tissue.

Section No. 3. This comprised ovary, chorion, amnion, and a few degenerate nonfunctioning decidua cells. The ovary was edematous. The vessels were large. There was a layer of fibrin that separated a few hyalinized, collapsed villi, from the substance of the ovary. There were no free intervillous spaces. The chorion was narrow, sclerosed and avascular. The amnion was well preserved. There were a number of cells, resembling decidua cells, embedded in the fibrin, along the surface of the ovary. *Diagnosis:* Ovary and placenta: the changes in the chorion and villi here resemble those in the area of decidua reflexa of a normal pregnancy at about this period.

Section No. 4. This comprised the broad ligament, vessels of the broad ligament, and chorionic villi. Along one side, the broad ligament showed an old, chronic inflammatory zone of scar tissue. Along the other, there was a reasonably well defined zone of decidua cells, covered by a layer of fibrin. Adherent to the latter was a zone of sclerosed, but cellular, and poorly preserved chorionic villi. These had poorly defined vessels. The intervillous spaces were, for the most part, free. A striking change was seen in the vessels of this broad ligament. They were enormously dilated, and were bordered by smooth muscle fibers that showed an extreme degree of hypertrophy and hyperplasia. There is also a striking hyperplasia and hypertrophy of all of the smooth muscle fibers of that surface of the broad ligament coming into close contact with the placenta. There was no ingrowth of villi or trophoblastic cells into the tissues of the broad ligament. *Diagnosis:* Broad ligament, showing increased vascularity, hypertrophy of muscularis, and decidua cell formation, with attached, nonfunctioning, sclerosed, chorionic villi; old, healed inflammation of the broad ligament; chronic inflammation of the broad ligament.

Section No. 5. This consisted of the broad ligament. This showed massive hypertrophy and hyperplasia of the muscle fibers of the blood vessels. There were signs of old, healed inflammation. There was one fresh hemorrhage into the loose, edematous stroma. *Diagnosis:* Broad ligament, showing hemorrhage, edema, and vascular hypertrophy.

Section No. 6. This section comprised the broad ligament, tube and placenta. This section of tube showed a chronic inflammatory reaction of the wall, with extension to the surface as a chronic perisalpingitis. Along one side of the outer surface of the tube, there was a zone of decidua and a layer of fibrin. Embedded in the latter were few degenerate sclerosed, non-functioning, chorionic villi. *Diagnosis:* Chronic salpingitis and perisalpingitis, with attachment of non-functioning placental tissue along the serosal surface of the thickened tube.

Section No. 7. This comprised the tube, broad ligament and chorionic villi. The tube, in this area, showed signs of old, healed salpingitis, with adhesions between the villi and consequent partial obliteration of the lumen. The lumen of the tube was covered with an uninterrupted layer of epithelium, but the adjacent connective tissue and smooth muscle that form the wall were sharply interrupted in one area. At this site, the wall of the tube was thin, edematous, and rich in large, gaping, blood sinuses, and clusters of decidua cells lay in the mucosa and in the adjacent wall. A single layer of epithelial cells was the only barrier separating these decidua cells from the lumen of the tube. There was considerable old blood, coagulated in and about this area, and functioning and non-functioning, sclerosed, chorionic villi were deeply embedded in both this decidua tissue and blood clot. Peripherally, this defect in the wall was partially filled with placental tissue, which became continuous at the surface of the tube with a layer of fibrin, decidua and villi covering the broad ligament. *Diagnosis:* Ectopic tubal pregnancy with extension of the pregnancy through the mucosa into the wall and with growth of the placenta both within the wall of the tube and along the adjacent broad ligament; old, healed salpingitis.

Section No. 8. This comprised the broad ligament and organizing blood clot. This section was taken in one of the thinnest portions of the placenta. The broad ligament showed a definite chronic inflammatory reaction in this area. There was considerable hemorrhage showing signs of organization. There were no decidua and no villi. *Diagnosis:* Broad ligament, showing chronic inflammation, organizing blood clot.

Section No. 9. This consisted of the broad ligament and placenta. This section, like the last, was taken in the thin portion of the placenta, and included its entire thickness. The broad ligament was bordered along one side by old, organizing bloodclot in which were embedded degenerating chorionic villi. The chorion was narrow and sclerosed. *Diagnosis:* Broad ligament, showing infarction of villi and organizing hemorrhage.

Section No. 10. This specimen was of the chorionic villi. This section was taken through the chorionic villi in the thickest portion of the placenta. The villi were healthy, mature, and showed normal vascularity. There was no suggestion of erythroblastosis. *Diagnosis:* Healthy chorionic villi, entirely free of disease.

Section No. 11. This section was the cord. This is a cross-section, taken through the cord, several centimeters from its placental attachment. The three vessels were well formed and bore a normal relationship to each other. The bordering tissue was quite regular in all respects. *Diagnosis:* Cord, showing no evidence of disease.

SUMMARY

From a review of the above descriptions and diagnoses, it is apparent that this represents an abdominal ectopic pregnancy, apparently originating within the tube. The development of the placenta had taken place principally within the wall of the tube and along the surface of the tube, ovary and broad ligament.

The tube and ovary were the seat of an old, healed inflammatory reaction in which the lumen of the tube had become partially obliterated. There was still a mild chronic inflammatory reaction in portions of the broad ligament.

The cord and the amnion and the chorionic membrane and chorionic villi adjacent to the attachment of the cord were healthy, mature and free of disease. The chorionic villi in this portion of the placenta were loosely attached to the broad ligament in the region of the tube and ovary. In this area, there were well developed intervillous spaces, bordered by chorion on the one hand, and by fibrin, decidua, and smooth muscle fibers of the broad ligament, on the other. There was a hypertrophy and a

hyperplasia of the smooth muscle fibers of the broad ligament, and of the vessels of the broad ligament, especially those in the immediate vicinity of the attachment of the placenta. The vessels here were large, dilated, and gave rise to broad, thin, endothelial lined sinuses, bordered by edema fluid, fibroblasts and decidua. Chorionic villi and nests of trophoblasts were found within the lumina of some of these thin-walled sinuses. The decidua, connective tissue stroma, smooth muscle fibers of the broad ligament, and the dilated and hypertrophied vessels represented the functioning portion of the maternal placenta. The remainder of the placenta was thin, and for the most part, non-functioning, and was composed of a compact layer of chorionic villi that showed degeneration and necrosis, fibrin, and bloodclot. This portion of fetal placenta was attached to portions of the broad ligament and pelvic peritoneum and to the surface of the ovary by dense fibrin, which showed very little attempt at organization. There was no evidence of malignancy.

COMMENT

There are several points of unusual interest in this case. First, the abdominal pregnancy itself; secondly, its probable origin from within the tube; thirdly, the ectopic decidua cell reaction along the surface of the broad ligament; fourthly, the striking hypertrophy and hyperplasia of smooth muscle in the immediate vicinity of the functioning chorionic villi,—which suggests a hormonal stimulus to growth, and lastly, the lack of invasion of the villi in areas devoid of decidua.

The course of the large intestine affected by thrombo-ulcerative colitis is relatively straight and the angles at the flexures approximate right angles. From a soft, pliable, thin walled, gently winding, and twisting tube, it has become a hard, inflexible, stiff, thick walled, straight pipe with a small lumen somewhat analogous in form and general appearance to a thick walled, tense sclerotic artery.

REGIONAL ILEITIS*

MANAGEMENT OF COMBINED INVOLVEMENT OF DISTAL ILEUM AND DISTAL COLON

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DESPITE the contention of Maingot[1] that Combe[2] is to be credited with the first description of terminal ileitis, the present concept of the disease is based upon the paper published by Crohn[3] in 1932, after observing fourteen cases. These were clinically and pathologically similar. In each the involvement was of the terminal ileum by a non-specific granuloma with predisposition to the formation of internal and external fistulas and finally to stenosis of the affected and involved segment.

Harris, Bell and Brunn[4] in 1933, reported jejuno-ileitis as a similar disease; Brown, Bargen and Weber[5] included both forms under the term regional enteritis. Colp,[6] in 1934, described another form in which the small bowel involvement was accompanied by an inflammatory, ulcerative colitis, with symptoms and course at variance with those of involvement of ileum alone.

At first, Crohn[7] was unwilling to accept this new disease as due to the basic condition described in 1932. Crohn based this unbelief upon the fact that in all the cases originally described, the lesion involved the terminal eight to ten inches of the ileum, ending abruptly at the ileocecal valve. In addition, he cited examples in which a diseased ileum had been anastomosed to a healthy colon, and the colon had remained free of involvement even after sixteen years. However, he reported nine cases of a combined type of regional inflammation, an involvement of the small and large intestines, the ileum reacting as a granuloma, the colon reacting as an ulcerative and hyperplastic colitis.

Such a combined type of disease, according to Crohn, is not usually sequential but synchronous. Sometimes, however, it seems as if the disease had been primary in the ileum and spread to the colon secondarily.

It must be pointed out that a severe ulcerative colitis may extend to involve the terminal ileum. Such a condition has been well shown by the work of Henke and Lubarsch,[8] Bargen, Buie and Rankin,[9] as well as Klemperer.[10] They place the incidence of such an extension at about 25 per cent of all cases of colitis.

All of these writers are agreed that although extension may occur from the colon to the ileum, the extension is by backwash through the ileocecal valve, and the process in the ileum in ulcerative colitis is destructive and denuding, as in the colon, and not hyperplastic and granulomatous, as in primary ileitis. The clinical picture is one of colitis and not of ileitis.

Crohn gives the typical description of the combined form of primary ileitis and colitis in his later paper which is quoted as follows. "An ulcerative, granulomatous

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inflammation of a severe type involves the terminal ileum, and scattered and interrupted segments of the colon, or the contiguous cecum and ascending colon. The clinical picture is essentially that of an ileitis which must be considered the dominating factor and as contributing to the main clinical features. The onset is usually stormy and febrile with high temperature, abdominal pain, and a low grade diarrhea with from two to three movements a day. The course soon becomes chronic, debilitating and emaciating. The entire pathologic process may subside after many months, with complete restoration to health, or resection of the ileum and the contiguous ascending colon may be followed by a subsidence of the colitis and cure. Resection of the colon is not mandatory, removal of the diseased ileum, not in part, but in whole suffices to allow complete subsidence of symptoms. The involvement of the colon in the picture may be seen at the exploratory operation, or may be reported roentgenologically by barium enema, or it may be seen in the later stages through the sigmoidescope when the extenion has proceeded to the terminal colon. The differentiation from primary ulcerative colitis is made on the basis of the lesser severity of the colonic involvement, the mild degree of diarrhea at the onset and the presence of the predominant lesion in the ileum, namely, a mass in the right iliac fossa, abdominal cramps and eventual ileal stenosis. Fistula formation is rare in this combined form. The etiology is not known."

Fig. 1. Barium enema showing appearance of involved terminal ileum and distal colon; after exclusion.

Fig. 2. Barium enema showing fistulous tracts between involved areas in distal colon; before exclusion.

Ginsburg and Garlock,[11] in a resume of the decade of experience at Mt. Sinai Hospital, state that of the three similar lesions included under the name regional enteritis; i.e., jejuno-ileitis, distal ileitis, and the ileocolic form of right-sided migratory colitis, the first is definitely nonsurgical. They also show that exclusion without resection gives satisfactory results in distal ileitis. Certainly none of those who have reopened the abdomen to resect

the formerly massively infiltrated segment can have failed to be impressed with the subsidence of infiltration and adherence following exclusion.

FIG. 3. Resected specimen in case of combined involvement of distal ileum and distal colon.

In the case here reported, the retroperitoneal ileosigmoid fistulas did not heal on exclusion, the cecum apparently became involved after the exclusion, and the disease persisted in the rectosigmoid long after resection of the original involved ileum and exclusion of the rectum.

CASE REPORT

Mr. E. C., a white male, age twenty-one, No. 40-27569, was admitted to the Cook County Hospital on May 14, 1940. At this time he was complaining of vague intermittent pains in the abdomen for seven days. These pains had localized to the right lower quadrant five hours prior to admission. There was no associated diarrhea or constipation. He did vomit five times during the day before admission. Essential physical findings at this time were limited to the abdomen, and consisted of tenderness in the right lower quadrant, with no abdominal palpable mass. Occasional rushes and tinkles were heard. Rectal examination was negative. The white blood count was 13,600; hemoglobin 90 per cent; urine negative, temperature 98.8°F.; pulse 80; respirations 20. A diagnosis of acute appendicitis was made and operation performed. The note made by the surgeon follows: "Appendix encased in a mass of omentum about the size of an orange. Free fluid present in the peritoneal cavity, the *terminal ileum much inflamed.* The abdomen was opened with a McBurney incision, the mass noted as above and the abdomen closed without removing the appendix." The postoperative course was uneventful and the patient left the hospital on the eighth postoperative day.

Ten months later, March 27, 1941, he came under our care on readmission to the Cook County Hospital, No. 41-17104. The history on this admission was as follows: He stated that he had been ill for over a year with vague lower abdominal crampy pain. In addition he had had postcibal pain, referred to the right and left lower quadrants, regardless of the kind of food eaten. He had lost thirty-five to fifty pounds in the last six months, and had become gradually weaker and emaciated. In the last month, he had had a watery diarrhea, with three to four movements a day. The stool had contained no gross blood. The abdomen was scaphoid, no rigidity or masses palpated. Tenderness was present on deep palpation in the midline between the xyphoid and pubes. Bowel sounds were hyperactive with occasional rushes and borborygmi. The prostate was firm and no other mass was palpable.

The provisional diagnosis was ulcerative colitis, to consider enteritis. After the previous chart was obtained and it was seen that in the previous operation the inflamed terminal ielum was noted, with a right ileal mass, the diagnosis was thought to be regional enteritis. The stools examined repeatedly contained no blood. Proctoscopy showed at 10 cm. a hard, irregular friable ulcer involving the left posterior quadrants. A punch specimen was taken for biopsy. Urine was negative; chest plate was negative. After a barium enema the following was reported: "There is a rigid, irregular filling defect of the distal portion of the sigmoid colon, with multiple sinus tracts extending from this point. The lumen is markedly narrowed. The terminal ileum is also markedly narrowed and irregular, with sinus tracts arising from this area, some of which apparently join the tracts arising from the sigmoid colon, thus giving rise to fistulous tracts between the ileum and sigmoid. The findings are diagnostic of a regional enteritis."

Preoperative management included transfusions of blood, vitamins, adequate diet and

hydration. At operation on April 18, 1941, the findings were as follows: Twelve to fifteen inches of the terminal ileum, were involved in a densely adherent mass with the pelvic colon and rectum. The cecum was uninvolved as was the proximal ileum and jejunum. Many almond-sized firm glands were in the mesentery. The serosa of the involved ileum was still somewhat shiny, but the wall was many times thickened. A right rectus displacing incision was made. The proximal loop of healthy ileum was brought six inches out the upper end of wound, and put through a slit in the muscle. The wound was closed in layers with interrupted silk. A full thickness free skin graft from the right thigh was put around three inch loop of ileum as a sleeve, and sutured to the abdominal wall. The donor site was closed with interrupted silk. Two days later the ileostomy was opened and a catheter placed in the proximal limb. The limbs were divided on the tenth day. The patient made an uneventful recovery. On May 13th, sigmoidescopic examination revealed a friable ulcerated mass at 10 cm. on the left posterior area. From this there were several fistulous tracts, their bases easily evident, from which might be expressed a thick yellow drainage. This looked less acute and inflamed than when we saw it previously, however. Biopsy of area showed an actively inflammatory lymphoid follicular polyp; there was no evidence of malignancy. Biopsy of abdominal lymph node revealed non-specific hyperplasia with marked proliferation of the reticulum in the secondary follicles.

The patient left the hospital on May 14th, gaining in weight and strength and with his ileostomy functioning well, retained by a colostomy bag.

On October 1st, he was again admitted to the Cook County Hospital, No. 41-55670, having been told to report at this period for further examination and treatment. During the interim he had, in a period of six months, gained thirty pounds, and had had no further abdominal cramps. He had had some mucoid, blood tinged drainage from the rectum during this time. Essential physical findings were the ileostomy openings at the apex of the skin tube in the right mid abdomen, and the rectal induration with the seromucopurulent drainage. The abdominal skin was normal, ample reward for the trouble in making the skin tube.

Sigmoidoscopic examination revealed that between 10 and 20 cm. the mucosa was ulcerated and contained openings of several fistulous tracts. The mucosa was reddened and hyperemic. The impression was that of an ulcerative proctitis secondary to enterofistulas from adjoining regional enteritis.

To continue the defunction of the rectum after the segment of ileum was resected, a loop colostomy of the sigmoid was performed under intercostal block on October 6th; this was opened on October 31st. A Frei test was negative at this time.

On October 20th, under spinal nupercaine, a vertical incision was made excising the ileostomy. The terminal twenty inches of ileum, which had been indurated and sausage-like at the previous operation, was now small, thickened and matted together. The cecum was thickened and small. The mesentery of the ileum had glands enlarged, almost to the root. The terminal ileum was twisted on its self, and attached to the cecum, suggesting internal fistulous tracts between the ileum and cecum. The rectosigmoid was firmly attached to the posterior pelvic wall. No obvious communications were seen between loops of terminal ileum and rectum. A right hemicolectomy, including the distal twenty-four inches of ileum was performed. An isoperistaltic ileotransverse colostomy with two rows of interrupted silk sutures was performed. Layer closure, without drainage, with interrupted silk was done.

A description of the resected specimen, by Dr. A. B. Ragins follows: "Resected portion of an ileostomy wound closed by black silk, communicates with proximal loop of ileum measuring 15 mm. in diameter, also with the terminal portion of the ileum which measures 35 cm., the cecum, ascending colon and proximal part of transverse colon; 15 cm. from the distal ileostomy opening, the loop of the ileum shows a U-shaped defect. The top of this defect is composed of a dense fibrous tissue. The terminal 14 cm. from the ileocecal junction medialwards shows a marked thickening of the wall, the serosal aspect is darker purple red than the purple red of the normal mucosa. The wall is up to 4 mm., light grey. The mucosa is edematous, attached to the underlying tissue and is slightly granular. The remaining loop of ileum extending to the distal ostium of the ileostomy wound, light pinkish grey, the wall 2 mm. thick, the mucosa pink grey and folds

are distinct. At the top of the U-shaped defect there is formed a small traction diverticula measuring 12 mm. in length. But this diverticulum does not communicate, although it is firmly adherent by fibrous connective tissue bands to the distal most portion of the ileum which forms the other prong of the U-shaped defect. The lymph nodes of the mesentery are up to 12 mm. in diameter, light purple in color. The follicles of the large bowel are distinct and the mucosa of the bowel is studded with numerous pinpoint to pinhead sized hemorrhages. The appendix measures 5 x 4 x 6 cm. and on section through the tip, the lumen is markedly narrowed. Microscopic: Specimen of terminal ileum at the site of construction reveals the mucosa to be superficially ulcerated and markedly infiltrated with numerous round cells, which extend to the thickened submucosa. The muscularis propria is but moderately infiltrated by round cells, and the subserosa shows marked infiltration. This microscopic picture is in keeping with the diagnosis of regional enteritis. Section of a lymph node shows a non-specific inflammatory hyperplasia."

Following this procedure, the patient made an uneventful recovery and was discharged on November 12th, having four to five watery stools daily through the colostomy. On November 10th, sigmoidoscopic examination had shown considerable scarring of the mucosa, and at 12 cm. on the right posterior there was seen a definite fistulous tract covered by a large inflammatory polyp. Above this up to the sigmoid there was irregular scarring and several small inflammatory polyps. The mucosa of the sigmoid was fairly normal.

On March 30, 1942, he was again examined. He entered the hospital desirous of having the colostomy closed. He had been free from pain and of discharge from the rectum since the last operation, had continued to gain weight, and now weighed more than prior to his illness. Sigmoidoscopic examination revealed a thickened rectal mucosa and many inflammatory polyps. Biopsy of the largest of these revealed only non-specific inflammatory hyperplasia. A barium enema passed freely through the distal colon and out the distal limb of the colostomy. It was decided that the sigmoidostomy could be closed. On April 13th, under 125 mg. of 10 per cent procaine intrathecally, the stoma was excised and the skin freed. The fascia was exposed all around, the peritoneum not opened. The serosa of the bowel was cleared of fat and epiploic appendages, and closed transversely with two rows of interrupted silk. This was then reduced under the fascia, and the fascia imbricated with silk, and the skin closed. Sulfathiazole, 30 gr., were placed in the wound.

The patient left the hospital on April 28th, having watery to soft stools. The colostomy seemed to be closed. He was instructed to report monthly for recheck. After two months, the colostomy had begun to leak at intervals. Another attempt at closure was made July 31st. He returned in November, 1942, leaking from a small sinus again, and was admitted in January, 1943, and reoperated upon February 2, 1943.

Under spinal nupercaine, a longitudinal incision was made lateral to the fistula and the peritoneum opened by intention. The sigmoid loop was cleared and the fistula followed to the abdominal wall. The tract was excised and the bowel closed with two rows of interrupted silk, at right angle to the longitudinal axis. Inasmuch as several efforts had been made to close this fistula, it was feared that the lumen would be constricted, and a primary sigmoidosigmoidostomy was effected between the afferent and efferent limbs of the sigmoid. This was done with two rows of interrupted silk. Sulfanilamide, 60 gr., were placed in the abdomen, and the wound closed in layers without drains. The wound healed by primary intention, and the patient left the hospital, following an uneventful recovery, on February 15, 1943.

At the time of this patient's discharge, he was having normal bowel movements through the rectum, and had gained over fifty pounds from that of the entrance weight on March 27, 1941. Sigmoidoscopic examination showed at the old fistulous sites evidence of granulation, and reddening of the mucosa, but no fistulous tracts or openings, and no discharge into the lumen of the rectum.

This case is of interest from the standpoint of the length of time necessary to effect a cure, almost two years. We became discouraged at times with the course, and with the necessity of having to do graded, stage operations. We believe now that if we had originally opened the peritoneum to close the subsequent sigmoid fistula, the course of treatment would have been shortened, but we were hesitant to do this until the last attempt. The probability is

that this patient had now so much resistance to his intracolonic organisms, that the peritoneal cavity could easily have tolerated almost any intra-abdominal primary procedure.

This patient has been followed at weekly intervals, and there is no further evidence of any more fistulous tracts, the abdominal fistula is closed and the abdominal wall well healed. He is now in the army, having passed the physical examination necessary for induction.

SUMMARY

1. The close relationship between terminal ileitis as first so named by Crohn, jejuno-ileitis, ("skip" lesions) and combined terminal ileitis and colitis is briefly discussed. It is noted that the first type in which fistula formation is commonest, is now believed to be best treated by exclusion without resection.

2. A case of terminal ileitis is reported in which ileosigmoid fistula formation followed exploratory operation only. The ileal granuloma, but not the fistulas, and the rectosigmoid ulceration only slightly regressed after exclusion.

3. The operative stages in defunctioning and excision of the involved areas were: (a) end ileostomy, (b) sigmoid colostomy, (c) right hemicolectomy, including involved ileum, (d) closure of the sigmoidostomy, involving sigmoido-sigmoidostomy.

4. The treatment of such conditions is resection; to obtain a satisfactory result by graded operations may take up to two years; the period in which new lesions, if they are to occur, may be expected.

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SUBSCAPULAR OSSIFIED HEMATOMA*

CASE REPORT

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A CIRCUMSCRIBED collection of effused liquid or clotted blood in the tissues, their interstices, in serous or non-serous cavities constitutes a hematoma. In other words, a hematoma is a confined hemorrhage in which clotting may or may not have taken place. Although restraining influences are admittedly present, the implication that the effusion is always self-limiting or inherently controlled is contradicted by the frequency of local recurrence and regional extension.

Liberally viewed, hematomas range in size from pin-point dimensions (petechiae) to ponderous volumes. Ecchymoses differ from petechiae in being larger and confined to the skin, but they are actually hemorrhagic effusions.

Classification according to source, physical features, anatomic site, pathologic characteristics, and period of existence tends to simplify description. Therefore, a hematoma may be referred to as acute, postoperative, diffuse, subcutaneous, infected, or in analogous applicable terms. The acute stage is arbitrarily considered to last three weeks and the chronic indefinitely thereafter.

Hemorrhagic accumulations in serous cavities, although hematomas in fact, are designated hematoceles, hematocysts and blood cysts, or they are given appellations of specific localization, namely, hemothorax, hemoperitoneum, hemarthrosis, and equivalent titles. In some instances, particularly when liquefaction has taken place, they are termed hydromas or hygromas.

Hematomas appear spontaneously in consequence of dyscrasia, from the dissolution of a degenerated blood vessel and as an effect of constitutional disease. More often, however, they arise primarily from trauma, which is usually of a contusing or crushing nature, or secondarily from extrusion of a thrombus, the release of an insecure ligature, the failure to seal a venopuncture or from capillary leakage. Conspicuous sources of hematomas currently are attributable to warfare: the disruptive impact of fragmented projectiles, the division of vessels by penetrating missiles, the effect of vigorous traction induced by centrifugal force within the radii of powerful explosions, the bruising, rending and torsion of structures consequent to obstacle course training and the severe crushing injuries sustained in vehicular accidents. Fracture and dislocation of the subjacent bones are liable to occur when violence is a factor and a hematoma may form from the site of fracture or from the forcibly separated contiguous tissues.

The fate of hemorrhagic accumulations is variable. When small and uncomplicated, they are accompanied by few if any symptoms and tend to resolve quickly. Treatment then is a minor consideration. Hematomas of larger proportions, however, are usually symptomatic although they may be the hidden complement of more obvious and serious lesions. Where injury is extensive, and particularly in deep lying regions, the hematoma may be completely obscured. The diagnosis then will depend upon the pathognomonic syndrome produced, as, for example, is the case in extradural hematoma, or upon subsequent developments as shown in

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the succeeding case report. Contingent upon location, associated conditions and complications, the treatment may be simple or involved.

Fig. 1. *X*-ray plate showing subscapular ossified hematoma compressing ribs.

Collections of blood which are not successfully evacuated pass through phases similar in some respects to the process of tissue repair, i.e., resolution, fibrosis, suppuration or, in rarer instances, ossification. Resolution may be delayed or interrupted. Failure in absorption results in the persistence of an hydromatous cyst. Because of the propensity of hematomas to become infected, a considerable number progress to abscess formation. Aspiration or excision, or incision and drainage are indicated, respectively, in either of the latter eventualities.

Ossification, an abstruse phenomenon, occurs with greatest frequency in hematomas about the elbow joint and in the thigh. Often manifested as a later transition product, calcium deposits have been demonstrated roentgenographically early in the third week following injury; and it is presumed that they were present for a brief interval, at least, before their acquisition of an opaque quality. Serial *x*-ray pictures of ossifying hematomas show a progression from flocculated calcium islets to complete replacement by lamellations of compact and cancellous bone. Eventually, the inflammatory barrier found at the periphery of hematomas is converted into a neomembrane, grossly resembling periosteum and serving as a capsule at the circumferential limits of ossification.

The primary objective in the treatment of ossified hematoma should be prevention. Experience has shown that aspiration and evacuation of simple hematomas are inconstant but likely sources of infectious contamination. Relative rest, passive motion, heat, diathermy, and massage are of benefit to an indeterminate number of patients in whom local calcification is of recent origin. In others, surgical excision may offer the only means of relieving disability. Recurrences after operation are fairly common either because ossifica-

tion was incomplete at the time surgery was instituted or because of the trauma incidental to operation.

case report reveals the advanced degree of ossification that may ultimately take place in a hematoma and the signs of

FIG. 2. *X*-ray plate showing ossified hematoma and diodrast injected vein.

The optimum time for surgical intervention has not been clearly defined. Too early removal may result in recurrence or extension of calcification; too late, in permanent pressure damage to adjacent vessels and nerves. Even though successful extirpation has been accomplished within three months of injury, it is perhaps more prudent to recommend waiting a year before making an attempt to excise an ossified hematoma, provided, of course, that relief from neurovascular symptoms is not indeed more urgent. The appended nerve degeneration that may result from pressure if operation is too long deferred.

CASE REPORT

R. W. D., No. 47192, a white army private, was admitted to the Station Hospital, Camp Joseph T. Robinson, Arkansas, March 20, 1943, complaining of a "lump in the right shoulder with numbness and tingling of the right hand."

The family history was negative for hematopathology and details of the past history were irrelevant.

The present illness was initiated twenty-two years previously when a severe crushing injury to the right shoulder was sustained in a fall beneath a thrown saddle horse. Treatment was promptly instituted and a plaster jacket worn for the succeeding three months. A similar period was devoted to physiotherapy in an effort to relieve persistent numbness of the right hand. Thereafter a cock-up splint was applied to the forearm and hand which were thusly supported for one and one half years. Sensory phenomena persisted, but the presence of a localized swelling above the clavicle and in the axilla was not discovered until several years after injury. The tumefaction was not believed to have increased in size although its presence was observed for fourteen years.

Fig. 3. Ossified hematoma specimen not magnified.

Physically, the patient was apprehensive, fair skinned, slightly pale and well nourished. The right hand was continually supported in the opposite palm and the arm was maintained at a twenty degree angle of abduction. The vertebral border of the corresponding scapula was rotated posterolaterally. The thorax was symmetrical, but there was an ovoidal fullness of the right supraclavicular fossa. An arterial pulsation was visible and palpable for a distance of approximately 7 cm. transversely between the clavicular and sternal divisions of the right major pectoral muscle. These pulsations corresponded with the heart rate and were easily compressible. They disappeared when the arm was elevated above a right angle. A local bruit was not present, although at the cardiac apex there was a soft, untransmitted systolic murmur. Pain, which radiated along the ulnar surface of the arm and forearm was induced by elevating the arm above shoulder level and by elevating the chin forcibly while the head was rotated toward the side of the lesion. An obliquely directed ovoidal, non-tender, solid, regularly surfaced tumor, movable vertically, medially, and laterally within narrow limits, occupied the right subscapular, axillary, and supraclavicular fossae. The overlying skin was not inflamed nor was it adherent to subjacent structures. Forceful pressure mediosuperiorly in the depths of the axilla induced local pain and increased the tingling sensation along the ulnar nerve distribution of the forearm and hand.

Neurologically, there was hypesthesia of the dorsal and ventral aspects of the medial half of the right fourth and total fifth fingers. The sensory impairment extended over both surfaces of the hand and forearm to approximately 6 cm. above the ulnar carpal joint. Segmental involvement of the eighth cervical and first dorsal nerves indicated that elements of the ulnar and median nerve branches were involved in the pathologic process through compression of the medial cord of the brachial plexus. However, there was no loss of reflexes and no muscular atrophy. Likewise, vascular phenomena were not evident, there being no ischemia, cyanosis, mottling, or edema.

Stereoscopic *x*-ray plates showed a large, irregular, calcified mass measuring approximately 14 by 6 cm. located between the scapula and the thoracic wall. The mass was sharply demarcated from the surrounding soft tissues and was not attached to any bony structure, although the upper four right ribs were flattened against the lateral lung surface, thus narrowing the costal cage, and the scapula was moderately displaced backward. Impression: Calcified hematoma.

A tangential view after the injection of 30 cc. of diodrast into the median basilic vein revealed satisfactory visualization of the basilic and axillary veins. The axillary vein was displaced anteriorly and 1.5 cm. above the calcified mass.

Other pertinent laboratory studies were of no assistance either in the diagnosis or treatment.

On March 30, 1943, the tumor was removed under nitrous oxide-oxygen-ether anesthesia through an inverted J incision made from the level of the deltoid tubercle of the humerus and continued high over the central anterior aspect of the deltoid muscle medially to the acromion region and lateromedially downward to the fifth right interspace where it terminated 4 cm. from the midclavicular line. The skin flaps were dissected upward, medially and laterally to expose the anterolateral aspect of the pectoralis major and the lateral border of the latissimus dorsi muscles. The pectoralis major muscle was separated along the course of its fibers, between the clavicular and sternal portions, to the bicipital groove at which point the sternal segment was divided 2 cm. medial to its tendonous insertion. The sternal part of the muscle was then dissected medially to expose the axilla, pectoralis minor muscle, and costocorocoid membrane. Incision of the costocorocoid membrane permitted the insertion of the right index finger mediolaterally beneath and close to the posterior surface of the minor muscle. Upward and downward motion of the finger freed the muscle from subjacent structures to its insertion on the corocoid process. The suspensory ligament (fascia) of the axilla was incised at the lateral margin of the minor muscle which latter was then divided between hemostats

FIG. 4. Sagittal section of ossified hematoma not magnified.

3 cm. from its insertion. The inferior section of the pectoralis minor muscle was retracted downward and medially to expose the subclavian and axillary regions. The tumor was then clearly outlined, beneath a layer of fibroareolar tissue, fully occupying the subscapular space and projecting into the axillary and supraclavicular fossae. A tributary of the dorsal scapular vein appeared anteriorly, having penetrated the center of the tumor, and passed obliquely upward into the lateral thoracic vein. It lay between the capsule of the tumor and the more superficial areolar tissue. After severing this venous tributary between hemostats and ligating its terminal ends, the tumor was exposed by scissors dissection. Cleavage was obtained with difficulty and freeing of the mass in the subscapular depths by scissors and digital maneuvering was time consuming and toilsome. However, bleeding was minimal and of a capillary nature. After the tumor was extricated, the divided muscles were united to their respective tendonous insertions with interrupted, mattress, cotton sutures. Three gutta-percha drains were passed from the subclavian, subscapular, and axillary fossae through a stab wound in the skin and subcutaneous panniculus. The stab wound lay in the posterior axillary line opposite the fourth right interspace. The skin was closed with vertical, mattress, cotton sutures.

Grossly the mass was inclosed in a neomembrane. The specimen consisted of a heart-shaped tumor that weighed 390 Gm. It measured 13 × 11.5 × 5.5 cm. Adherent to its surface were numerous fatty tabs and some fibrous tissue. Several areas of hemorrhage were visible within the capsule wall. The consistency was hard and rock-like. On cross section the mass presented a mottled appearance, apparently consisting of rather dense cancellous bone with areas of intermingled fatty and red marrow. Considerable blood-tinged fluid escaped from the cut surface.

Microscopic section showed an encapsulated bony mass. The capsule was made up of pale pink-staining acellular connective tissue mingled with a small amount of fat. The surface of the bone was fairly smooth, but did not show a well defined periosteum. The cancellous bone in the central portion showed well formed, typical, bony trabeculae. No unusual amount of bone formation or destruction was evident. Between the bony trabeculae a typical marrow structure was evident. In some areas this consisted solely of fat while at other points the fat was interspersed with large areas of myeloid cells. These cells included megakaryocytes, granulocytes in all stages, and cells of the erythroblastic series. Considerable numbers of red blood cells were also seen at some points. Diagnosis: Ossified hematoma.

Progress, despite primary healing of the wound and the reasonably prompt return of function of the extremity, was trammeled by an inordinate apprehension of paralysis, the result of psychologic conditioning. Repeated warnings and advice against operation by previously consulted physicians, in whom the patient had supreme confidence, covertly inhibited recovery through the expectation of an unfortunate consequence. Therefore, the hospitalization period was prolonged for the purpose of allaying fear through the medium of objective substitution and physiotherapy. The patient was discharged to duty on June 14, 1943. Function was completely returned, although there was a tendency to hold the distal phalanges of all digits in partial flexion. The neurological findings twenty-six days after operation were essentially unchanged, but the numbness and tingling were relieved. *X*-ray examination on August 3, 1943, four and one-half months after operation, showed no evidence of recurrence.

COMMENT

In the case reported an ossified hematoma of long standing was successfully removed. Its location, clinical manifestations, and histologic characteristics were unique.

The deformity of the ribs and the displacement of the scapula were curious findings suggesting a peripheral deposition of calcium which, as the mass increased in volume, compressed the relatively less fixed adjacent structures. Early involvement of the medial cord of the brachial plexus was assumed from the history of numbness which persisted from the time of injury. Pain and tingling were of more recent origin and led to the conjecture that a slowly progressive peripheral extension had further involved the nerve

trunk and was responsible for mild, but increasing, vascular symptoms. A scalenus anticus syndrome was simulated.

The histologic structure resembled bone marrow, the apparent hematopoietic function of which was not satisfactorily explained. It was not considered of compensatory nature because anemia did not follow operation.

Adequate exposure and facility of approach, without the production of a disabling scar, was accomplished by a technic similar to that used for clearing the axilla in the process of radical mastectomy. The simplicity with which the subclavian, axillary, and subscapular regions can be exposed by the technic described warrants its recommendation as an approach to surgical lesions in those locations.

Immediate relief from the neurologic manifestations was not expected in view of the long history and the probability of nerve degeneration. Eventually, however, complete recovery is anticipated.

THE major complication in these cases (of regional ulcerative colitis) is colonic perforation. This may necessitate temporary colostomy proximal to the lesion with subsequent resection, anastomosis if possible, and ultimate closure of the colonic stoma. It is well to allow several months to intervene between the several stages of this procedure.

ORCHITIS AS A COMPLICATION OF BRUCELLOSIS (UNDULANT FEVER)

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NOT infrequently the urologist, and occasionally the general surgeon, is confronted with a patient presenting a swollen testicle with associated pain, tenderness and a temperature. These signs and symptoms are essentially those of an acute orchitis but the etiological agent must be determined if an accurate diagnosis is to be made and the proper treatment initiated. Experience with two patients having orchitis, in a series of seventeen cases of brucellosis, was such that their case reports might be of interest and aid to others who might have patients presenting similar symptoms.

CASE REPORTS

CASE 1. Mr. W. G., a thirty-six-year old, married, white farmer was admitted to the hospital on July 14, 1942. For at least the previous ten days the patient had had a temperature which rose at times to 104°F. The right testicle had been found swollen one morning at the onset of the fever and the swelling had increased slightly subsequently. Diarrhea had been present for two days; the bowel movements had numbered ten to fourteen daily. Nausea and vomiting had been present for the same period. Several times at home the patient had had severe shaking chills that lasted several minutes. Following these he had perspired profusely and his feet had become cold. Left frontal headaches had been present and a "stomach ache," which must have been lower abdominal cramps, was a complaint. Irritability, restlessness, malaise, and anorexia had been present according to the patient.

Inventory by systems was essentially negative except for frequent "nose bleeds" as a boy, occasional pain and bleeding with bowel movements, pruritus ani, and some loss of weight with the present illness. His past history included scarlet fever, smallpox, chickenpox, peritonsillar abscesses, gonorrhea, "sinusitis," and a fracture of the lower third of the right tibia.

Physical examination revealed a large, well developed, well nourished white male who was obviously ill. The blood pressure was 114 systolic and 74 diastolic; temperature, 99°F.; pulse, 88; respirations, 22. Positive findings included several carious teeth, atrophic tonsils, enlarged spleen, and an old scar on the lower third of the right leg. The right testicle was swollen (5 by 4 by 4 cm.) and tender to palpation. There was questionable swelling of the globus major of the epididymis.

The first hospital day was characterized by diarrhea and an oral temperature of 104°F. in the evening. Examination of the blood showed a white cell count of 9,500, a normal differential count, and a red cell count of 4,800,000 with 85 per cent hemoglobin. Treatment initiated by the attending physician included bed rest, liquid to soft diet, scrotal support, cold packs to the right testicle, and 3,000 cc. of fluids intravenously, containing 10 Gm. of sodium sulfathiazole (sulfathiazole had been given orally at home when patient had refused hospitalization).

On the morning of July 15th, a rectal examination was done under sodium pentothal because the patient complained of periodic rectal pain and it was thought a peri-anal abscess should be drained. No abscess was found. KaO Magma was prescribed for the diarrhea which continued; 2,000 cc. of intravenous fluids were given because of the profuse perspiration and diarrhea. Agglutination tests, which had been suggested by the writer, for paratyphoid fever A and B and typhoid fever were negative but that for brucellosis was strongly positive. The blood culture was reported as negative for twenty-four hours. Routine urinalysis, stained smear of urine

sediment, Kahn serology, and an *x*-ray film of chest were normal. Stool examination showed occult blood strongly positive, a few "pus cells," and no parasites. The sulfathiazole blood level was 8.62 mg. per cent. (Two days later it was 7.50 mg. per cent.) Sulfadiazine by mouth was ordered after the positive agglutination test for brucellosis was reported.

On the third day the temperature dropped from a plateau of 102°F. to 99°F. in the morning, but in the evening it was 103.4°F. A light diet was initiated during the day and oral intake of fluids was adequate for the diarrhea had ceased. Perspiration continued.

The remaining days in the hospital were characterized by normal temperature, absence of profuse perspiration and diarrhea, reduction of chemotherapy dosage, discontinuance of scrotal support and Kao Magma, and getting the patient up in a wheel chair the day before his discharge on July 20th, the sixth hospital day. The swelling and tenderness in the right testicle were both decreased.

An attempt was made to contact the patient six months later for a check-up on his subsequent course, but this was unsuccessful. The source of infection in this case was probably via unpasteurized milk from the family milk cow. The farmer's herd was to be tested for brucellosis by a veterinarian subsequent to hospitalization.

Case II. Mr. L. G., a thirty-two-year old, married, white automobile mechanic entered the hospital on December 2, 1942. A swelling had appeared in the right half of the scrotum five days previously. This swelling had increased as well as the associated pain. The patient had thought at first that he had a "rupture" from lifting something too heavy. A physician had been seen who told the patient that he had a "ruptured vein." On November 30th, the patient had had a temperature of 103°F. As a result sulfathiazole therapy had been initiated. The temperature on December 1st had been 102.8°F.

Inventory by symptoms revealed approximately three pounds of weight loss in the past year, recent night sweats, "light-headed feeling at times," frequent attacks of coryza, "bad tonsils," shortness of breath upon slight exertion, heartburn over precordium, occasional palpitation, "skipped heart beats," and lumbar backache since working in the garage.

Past history included measles, mumps, pertussis, chickenpox, scarlet fever, "heart trouble" when eight or nine years of age, and no history of venereal disease. He had been married thirteen years and his wife and two sons had been in good health.

Physical examination demonstrated an asthenic, well developed, well nourished white male who was not acutely ill. The blood pressure was 102 systolic and 52 diastolic. Temperature was 100.6°F.; pulse, 84; respirations, 26. Positive findings included hypertrophied tonsils; slightly coated tongue; and a heart, which was enlarged to the left, presented extrasystoles, and a possible soft systolic murmur. The right testicle was enlarged to the size of a small lemon (4.5 by 3.5 by 3.5 cm.) and was very tender to palpation. The epididymis did not seem to be involved.

Upon admission sulfathiazole was continued by the attending physician. The scrotum was supported and an ice bag applied to the right scrotum. Urinalysis and Kahn test were negative. Blood examinations demonstrated a white cell count of 9,000, a normal differential count, and a red cell count of 4,500,000 with 80 per cent hemoglobin. Memory of Case I, a brief clinical history, and the physical findings prompted the requisition of a brucellosis agglutination test by the writer. This proved to be strongly positive (1:320 dilution). Chemotherapy was then abandoned and specific vaccine therapy in graduated doses initiated.

The pain in the right scrotum, which radiated superiorly to the right inguinal region, and possibly to the lower part of the back, was absent after three days Soreness of the right testicle continued as did the swelling. There was profuse perspiration during the night and the patient complained of being weak in the mornings until December 7th. The temperature remained normal after December 4th and the patient was discharged on December 8th (sixth hospital day) considerably improved.

This patient was seen on January 20, 1943. There was no atrophy of the testicle. The only complaints were malaise, rather frequent headaches, and weakness upon exertion. He was doing light work and had not resumed his regular work. The source of the infection was determined after the patient was discharged. Unpasteurized milk had been purchased up to six weeks previous to hospitalization. Although the patient did not drink milk often, he had consumed some. The cow which had been furnishing the milk was tested for

brucellosis and found to have a "positive reaction." (Blood of the cow was sent to the State laboratory by a veterinarian and the agglutination test for brucellosis was found to be positive.)

INCIDENCE

Orchitis occurs as a complication in 4 to 10 per cent of all the cases of brucellosis according to various authors. Meakins[9] states that the system which bears the brunt of the disseminated lesions of brucellosis is the genitourinary tract with orchitis occurring in 10 per cent of the cases. Several cases have been described in the foreign literature but only two have been discussed at length in the American literature. Isaac[8] realized the significance of considering brucellosis in the diagnosis of orchitis when he described a case before a meeting of the urologists in 1937. Buckley,[2] another urologist, in presenting a case called attention to the increasing frequency of brucellosis and the likelihood of the presence of more associated genitourinary complications. Simpson[14] reported sixteen cases of painful swelling of the testes in his series of brucellosis cases around Dayton, Ohio. Four cases were reported by Hardy[5] in his series of 125 patients having brucellosis in Iowa.

ETIOLOGY

According to Hinman[6] there are a number of conditions responsible for an acute or chronic orchitis. The acute type may be an ascending type from the posterior urethra via the vas deferens and epididymis resulting in an epididymo-orchitis. Rolnick[11] believes this route of infection is most unlikely. Wesson[15] states that traumatic orchitis and that from strain have been shown to be misnomers. Such conditions result from trauma with subsequent ascending or metastatic infections.

Acute metastatic orchitis is usually a complication of infectious diseases. It has been reported in cases of mumps, diphtheria, scarlet fever, small pox, influenza, typhus fever, dengue, glanders, malaria, typhoid fever, and pneumonia. Gout and filariasis have also been reported. In addition, foci of infection may be a source. Such lesions include tonsillitis, furunculosis, osteomyelitis, acute articular rheumatism, dental abscesses, sinusitis, cholecystitis, and appendicitis. The organisms involved are pyogenic in character, usually Bacillus coli and staphylococci. The chronic type of orchitis is usually due to syphilis.

PATHOLOGY

Any organism in the blood stream may enter the testes and, if there is a lowered resistance locally, or an abundance of infective organisms, a foothold may be gained and an orchitis result. However, organisms that invade the general circulation are rapidly filtered out and destroyed so that as far as the testicle is concerned blood borne infections are relatively rare.

According to Boyd,[1] unilateral orchitis is more common than bilateral inflammation of the glands. The testicle is only moderately enlarged owing to the firm non-elastic tunica albuginea by which the organ is surrounded. It is for this reason the patient has excruciating pain. In metastatic orchitis the epididymis is rarely involved. There is no associated hydrocele with orchitis as with epididymitis because of the intact tunica albuginea.

Sharp[13] in his general review of the pathology of undulant fever comments on the fact that the literature is not yet adequate for a clear conception of the morbid anatomy of brucellosis. The chief contributing factor is the limitation of post-mortem material due to the low mortality rate. He cites the microscopic findings of a case of brucellosis reported by Rothenberg.[12] This seems to be the only pathological description in the English literature of orchitis complicating brucellosis and in this case the patient evidently did not have an orchitis as a clinical entity. It may be said the patient had been married thirty-two years without any offspring.

"The testes revealed complete aspermatogenesis with evidence of active interstitial orchitis and scattered areas of fibroid atrophy. Vacuolar degeneration of the epithelium was noted." Pathological descriptions are not to be found because the orchitis complicating brucellosis usually is a self-limited disease.

Hinman[6] states that, in general, acute inflammatory processes manifest themselves between, not within, the canaliculi of the testicle. Multiple foci of infection are seen. The tubules then degenerate and become filled with inflammatory exudate. Local areas of necrosis occur and become conglomerate. The process may go on to suppuration.

SIGNS AND SYMPTOMS

Severe testicular pain is usually the first symptom of orchitis for the reason given by Boyd.[1] Associated is tenderness and swelling of the organ, elevation of temperature, and, not uncommonly, nausea and vomiting. The scrotum usually becomes reddened. There is more prostration than in most cases of acute epididymitis. The pain may be referred to the back, the perineum, or upward along the spermatic cord according to Pelouze.[10] The globus major of the epididymis may become involved.

All of these signs and symptoms were seen in the two cases described and for this reason a patient may consult a urologist or a general surgeon although he has brucellosis. It is significant to note that the patient with brucellosis does not look as ill as his chart indicates. In other words, there is an appearance of well being inconsistent with the high fluctuations of temperature.

DIAGNOSIS

The combination of clinical history, physical examination, and laboratory studies in making a diagnosis is well illustrated here. A good history will elicit the systemic symptoms of brucellosis in addition to the course of local signs and symptoms.

Foshay,[4] who has been keenly interested in brucellosis, concludes that there are no laboratory tests for this disease which do not have serious limitations. He adds that there are no acceptable clinical criteria for the diagnosis of brucellosis, nor for its exclusion and, therefore, it is impossible to reach a clear cut decision concerning the validity of the tests, alone or in combination. Yet few diseases depend more on the laboratory for a diagnosis than brucellosis. Only in communities where the disease is known to exist can a diagnosis be made without a laboratory test and then only hazardously.

The specific agglutination test of the blood is of greatest practical value in the diagnosis. In the two cases presented the antigen of Brucella abortus was used. The blood serum from cases of brucellosis does not always agglutinate the antigen and that from patients without the infection may give positive agglutination tests in low titers. There have been patients with brucellosis with negative serum agglutination tests or positive only in low titers. However, patients with the disease usually give positive reactions in high dilutions (1 to 80 or better). Foshay[4] states that next to recovery of the Brucella organisms by culture, a high serum agglutination titer is the most reliable test. The test usually becomes positive by the second week of the illness. In men and inoculated animals there is cross-agglutination between the Brucella and Bacillus tularense of tularemia. However, the titer is usually higher for the specific disease.

TREATMENT

The therapy of orchitis is conservative in character; "watchful waiting" in the words of Pelouze,[10] for the infection usually subsides. Scrotal support gives considerable comfort. Local hot or cold applications and analgesics systemically are the basis of the treatment. Christopher[3] states that incision will rarely be necessary; the therapy should be directed toward the causal infection. Surgical in-

tervention is indicated if fluctuation and suppuration or necrosis develop. Drainage is then initiated for small areas of suppuration and orchidectomy if the area is large.

As for brucellosis, prophylactic treatment consists of pasteurization of the milk for human consumption. In addition, individuals in contact with livestock, as cattle and hogs, or fresh meat should protect themselves adequately. Vaccination of young animals and removal of those infected is said to prevent the dissemination of the disease. Prophylactic immunization has been reported. Specific therapy has not proved successful. Anti-serums, vaccines, brucellin, non-specific protein therapy, chemotherapy, and artificial fever have all been used with inconclusive results in patients with brucellosis. Sulfadiazine was used in the first case herein reported. In the second case an oxidized Brucella abortus vaccine (Foshay) was used. Symptomatic treatment utilizing vitamins, sedatives, laxatives, adequate fluid intake and nourishment is, of course, indicated.

PROGNOSIS

Orchitis is usually transitory and there are no serious after-effects when one of the Brucella is the causative organism. Occasionally, a suppurative orchitis or an epididymitis develops. Huddleson[7] states that there have not been any cases of sterility following the orchitis complicating brucellosis.

Although the mortality rate of brucellosis is about 2 per cent, according to vital statistics, the chronic invalidism of a patient with the disease is of some concern. Fatal cases result from acute overwhelming infections or the subsequent localized lesion in the meninges, brain, lungs, or heart valves.

COMMENTS

With the significant increase in the number of reported cases of brucellosis undoubtedly more of the complications of the disease will be in evidence. In Illinois, three times as many cases were reported in 1941 as in 1932. Only five cases had been reported from the county in the ten years preceding the six months in which the seventeen cases were observed. In the United States, the reported cases of brucellosis have steadily increased from twenty-four in 1925 to 3,427 in 1941.[7]

It appears that the genital tract attracts as many of the disseminated lesions of brucellosis in humans as it does in susceptible animals. As the localized genito-urinary lesion, orchitis may be the reason for the patient consulting the urologist or general surgeon. Such men should be ready to investigate obscure genital lesions for Brucella infections.

SUMMARY

Two cases of orchitis complicating brucellosis with additional remarks are presented.

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PENICILLIN

CASE REPORT OF A PATIENT WHO RECOVERED FROM PUERPERAL SEPSIS HEMOLYTIC STREPTOCOCCUS SEPTICEMIA

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A report of a patient recovering from a puerperal sepsis complicated by a hemolytic streptococcus (long chain variety) septicemia which failed to respond to the sulfa drugs but did respond to penicillin is indeed a therapeutic triumph which cannot be ignored and merits a place in the literature.

TABLE I

Date	Day Post-partum	Red Blood Cells, Millions	White Blood Cells	Hemoglobin		Blood Culture	Sulfathiazole (Blood) Mg. Per Cent	Urine	Transfusion	Sulfathiazole	
				Gm.	Per Cent					Oral	I.V.
4-10-43	1	....	.....	...	..	.....	.....	neg.	350 cc.		
4-11-43	2	2.2	7,600	9.5	65	.....	.....	....	.....	start	
4-12-43	3										
4-13-43	4	....	.....	...	..	.....	.....	....	.....	stop	
4-14-43	5										
4-15-43	6										
4-16-43	7	2.28	10,800	7.5	51						
4-17-43	8	1.7	6,200	7.5	51	taken	.....	....	.....	start	start
4-18-43	9	1.2	5,620	7.0	48	.....	.....	....	250 cc.		
4-19-43	10	....	.....	...	..	.....	13.2	neg.	.....	stop	stop
4-20-43	11	....	.....	...	..	.....	3.8	....	200 cc.		
4-21-43	12										
4-22-43	13	2.1	5,500	8.0	54	.....	.....	....	200 cc.		
4-23-43	14										
4-24-43	15	2.57	5,200	9.0	61						
4-25-43	16										
4-26-43	17	....	.....	...	..	no growth					
4-27-43	18										
4-28-43	19	patient discharged today									

CASE REPORT

The patient, a thirty-five year old white female, at term, gravida VII, para VI, entered Greenpoint Hospital on the evening of April 9, 1943, in active labor. At the time of admission the duration of parturition had extended fourteen hours. Three hours later a normal full term living female infant was delivered from the right occipitoanterior position. A complicated third stage of labor was anticipated since a history of two previous retained placentas, one fourteen years ago and one eight years ago, had been obtained. A bleeding third stage developed. Posterior pituitary extract (1 cc.) was given intramuscularly but proved ineffectual. Manual removal of the adherent placenta was completed under open drop ether anesthesia. Following this procedure the bleeding was controlled, not, however, until approximately a liter of blood had been lost. Replacement therapy consisted of a liter of 5 per cent glucose

in normal saline followed several hours later by a citrated blood transfusion, the latter finishing without noticeable immediate reaction. The blood pressure was recorded at 138/74, the pulse was slow and of good quality.

On April 11th, the second postpartum day, the patient had a severe chill with rise in temperature to 104°F. (oral). Sulfathiazole was started orally at this time. On the following day, a generalized urticaria developed to confuse the picture. The question of whether the reaction was from ergotrate, transfusion or sulfa drug remained undetermined. The consulting dermatologist pronounced the skin reaction a dermatitis medicamentosa and advised withholding therapy except supportive and routine postpartum care. Deprived of the best therapeutic measures the postpartum course became tempestuous until, on the eighth postnatal day, when the temperature rose to 106°F. (oral), it was decided to brave the consequences and once again institute the sulfa drug. The urticaria by this time had greatly diminished, hence, 5 Gm. of sodium sulfadiazine were given intravenously and sulfathiazole was started orally. Marked improvement in the patient's condition followed during the course of the next six days, the temperature falling to normal. A thrombophlebitis was noted on the fourteenth postpartum day in the left leg involving both the calf and thigh but appeared to be definitely subsiding. Discharge was granted on April 28th after five afebrile days; the hospital stay lasted nineteen days.

The patient was readmitted two days later complaining of chills, fever, lower abdominal and pelvic pain. The impression was that of a severe pelvic thrombophlebitis. The patient's course was notably stormy and response to therapy was nil. The organisms apparently had become sulfonamide fast. Oral and intravenous sulfa drugs were administered until the white blood count reached such a low level that it became inadvisable to continue. Frequent small tranfusions were given and convalescent serum (scarlet fever) was injected intravenously, but all to no advantage. The patient's condition became steadily worse until, in the opinion of those rendering therapy, little if any hope existed for recovery. The patient seemed moribund and terminus was expected momentarily. Five positive blood cultures were reported on May 13, May 18, May 24, May 29 and June 2, 1943. The cultures showed a long chain variety of Streptococcus hemolyticus. A large tender, readily palpable spleen presented itself. Petechiae in skin and mucous membranes were absent. No evidence of endocarditis was recognized. The lungs remained clear for the most part except for hypostatic râles in each base. The pulse was very rapid (132) and bounding; respirations (50) shallow and labored, the mental status befogged and speech delirious. Carphologia was present.

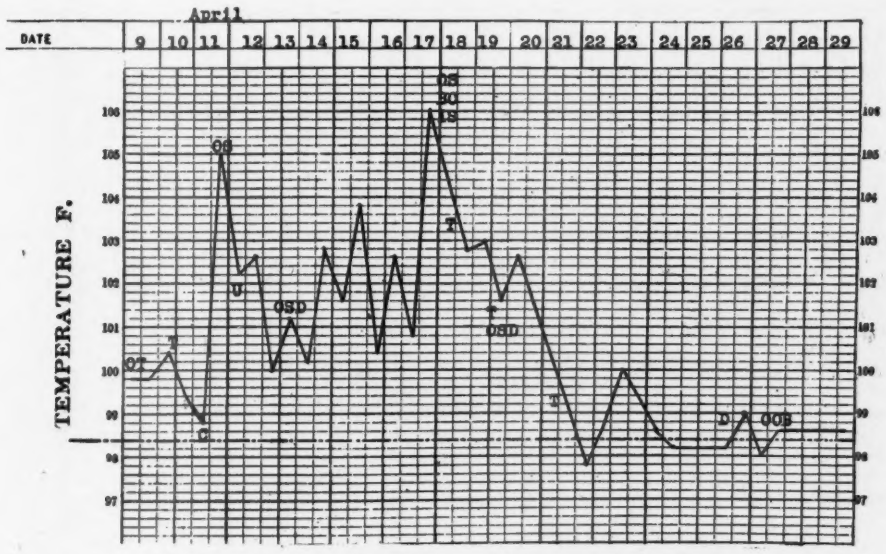

Fig. 1. OT, oral temperature; T, transfusion of citrated whole blood; OS, oral sulfathiazole; IS, intravenous sulfathiazole; OSD, oral sulfathiazole discontinued; U, urticaria; OOB, out of bed; BC, blood culture; C, chill.

Through the advice of Dr. H. J. Green, the administration of penicillin was begun; 38,800, fifth day 35,200, sixth day 35,200, seventh day 26,400. The first 74,200 units of

TABLE II

Date	Day Post-partum	Red Blood Count, Million	White Blood Count	Hemo-globin, Gm.	Differential			Blood Urea, Mg. Per Cent	Sed. Time 18 Mm.	Blood Culture	Trans-fusions
					Poly, Per Cent	Lymph, Per Cent	Mono, Per Cent				
4-30-43	21										
5- 1-43	22	2.31	29,300	8.5	86	14	..	..	20 min.	.....	.....
5- 2-43	23	....	.....	....	..	..	..	..	.....	.....	.....
5- 3-43	24	....	.....	....	..	..	..	..	.....	.....	.....
5- 4-43	25										
5- 5-43	26	....	.....	....	..	..	..	..	.....	.....	.....
5- 6-43	27	....	.....	....	..	..	..	..	.....	.....	.....
5- 7-43	28										
5- 8-43	29	....	.....	....	..	..	..	..	.....	.....	.....
5- 9-43	30										
5-10-43	31	....	.....	....	..	..	..	..	.....	.....	.....
5-11-43	32	2.57	3,700	7.5	50	50	..	..	15 min.	.....	100 cc.
5-12-43	33	....	.....	....	..	..	..	26	.....	.....	.....
5-13-43	34	....	.....	....	..	..	..	..	.....	positive	200 cc.
5-14-43	35	....	.....	....	..	..	..	..	.....	.....	.....
5-15-43	36	....	.....	....	..	..	..	..	.....	.....	250 cc.
5-16-43	37	....	.....	....	..	..	..	..	.....	.....	.....
5-17-43	38	2.56	4,500	7.5	54	46	..	..	.....	.....	.....
5-18-43	39	....	.....	....	..	..	..	..	.....	positive	250 cc.
5-19-43	40	....	.....	....	..	..	..	..	.....	.....	.....
5-20-43	41	....	.....	....	..	..	..	..	.....	.....	250 cc.
5-21-43	42	2.87	3,600	7.5	66	34	..	26	.....	.....	.....
5-22-43	43	....	.....	....	..	..	..	..	.....	.....	275 cc.
5-23-43	44										
5-24-43	45	2.62	4,200	7.5	77	21	2	..	.....	positive	250 cc.
5-25-43	46	1.89	3,800	7.5	63	37	..	..	.....	.....	.....
5-26-43	47										
5-27-43	48	....	.....	....	..	..	..	..	.....	.....	.....
5-28-43	49	....	.....	....	..	..	..	..	.....	.....	400 cc.
5-29-43	50	....	.....	....	..	..	..	..	.....	positive	.....
5-30-43	51	....	.....	....	..	..	..	..	.....	.....	.....
5-31-43	52										
6- 1-43	53										
6- 2-43	54	3.28	2,800	8.5	..	..	..	..	.....	positive	500 cc.
6- 3-43	55	....	.....	....	..	..	..	..	.....	.....	.....
6- 4-43	56	....	.....	....	..	..	..	..	.....	.....	.....
6- 5-43	57	4.14	3,550	8.0	77	23	..	..	.....	.....	.....
6- 6-43	58	....	.....	....	..	..	..	..	.....	.....	500 cc.
6- 7-43	59	2.69	4,650	8.5	70	28	2	..	.....	neg. 48 hr.	.....
6- 8-43	60	....	.....	....	..	..	..	..	.....	.....	550 cc.
6- 9-43	61	3.23	3,700	11.0	..	..	..	..	.....	neg. 48 hr.	.....
6-10-43	62	....	.....	....	..	..	..	..	.....	.....	500 cc.
6-11-43	63	....	.....	....	..	..	..	..	.....	.....	.....
6-12-43	64	3.82	5,700	10.5	68	32	..	..	over 1 hr.	neg. 48 hr.	.....
6-13-43	65	....	.....	....	..	..	..	..	.....	.....	.....
6-14-43	66	....	.....	....	..	..	..	..	.....	neg. 7 da.	.....
6-15-43	67	....	.....	....	..	..	..	..	.....	.....	.....
6-16-43	68										
6-17-43	69	3.20	5,900	9.5	54	46	..	..	.....	neg. 7 da.	
6-18-43	70	....	.....	....	..	..	..	..	1 hr.		
6-19-43	71										
6-20-43	72	....	.....	....	..	..	..	..	.....	.....	.....
6-21-43	73	....	.....	....	..	..	..	..	.....	neg. 4 da.	
6-22-43	74										
6-23-43	75										
6-24-43	76	4.32	5,600	12.5	62	36	2	..	1 hr.	.....	.....

42,400 Oxford units were given during the first twenty-four hours. This dosage was repeated on the second day. On the third day 40,600 Oxford units were injected, fourth day penicillin were conveyed by the medium of 5 per cent glucose in alternate solutions of distilled water and normal saline. The remainder was administered either intramuscularly or sub-

cutaneously in gradually decreasing amounts, dissolved in 2 cc. of normal saline. A total of

the patient's condition was decidedly improved. The fever began to subside, the mental

TABLE II (*Continued*)

Urine Findings						Convalescent Serum	Sulfa Drugs	Sulfa Titre Blood, Mg. Per Cent	Penicillin Oxford U			Comments
Sp. Gr.	React	Alb.	Cells	Casts	Cryst.				I.V.	Oral.	Sub-cut.	
1021	acid	neg.	occ.	neg.	neg.	.....	oral					
....	....	...	.....	.....	.....	.....	stop					
1017	acid	2+	15 wbc	.....	sulfathiazole							
....	....	...	.....	.....	.....	.....	.....	0.2				
....	....	...	.....	.....	.....	.....	.....	.....	.....	.....	.....	oxygen tent
1016	acid	tr.	3 wbc	occ. gran.	uric acid							
....	....	...	.....	.....	.....		oral					
....	acid	...	.....	.....	.....	.....	.....	6.2				
....	acid											
....	acid											
1013	acid	2+	many wbc	.....	sulfathiazole							
....	....	...	.....	.....	.....	.....	stop					
....	acid	...	.....	.....	.....	.....	oral					
....	....	...	.....	.....	.....	.....	oral i.v.					
....	acid	...	.....	.....	.....	.....	.....	0.7				
1022	acid	3+	occ. wbc	.....	sulfadiazene	.....	.....	8.0				
....	acid	...	occ. r and w	gran.	sulfadiazene	.....	stop	10.4				
1005	acid	2+	many r and w	hyalin								
1011	acid	2+	r and w									
1017	acid	2+	.....	gran. hyalin	.....	100 cc.						
1016	acid	3+	many r and w	gran. hyalin	.....	100 cc.						
1018	acid	4+	few r and w	gran.	.....	100 cc.						
1019	....	...	.....	.....	.....	100 cc.						
1012	acid	3+	many r and w	gran.	.....	.....	intra-venous	....	.....	.....	.....	patient looks moribund
....	....	...	.....	.....	.....	.....	.....	....	10600			
1007	acid	3+	few rbc	gran.	.....	.....	.....	....	31800	.....	.....	definite improvement
....	....	...	.....	.....	.....	.....	.....	....	31800	10600	.....	afebrile
....	....	...	.....	.....	.....	.....	.....	....	.....	50330		
1005	alk.	1+	occ. wbc	occ. gran.	urates	.....	.....	....	.....	48500		
....	....	...	.....	.....	.....	.....	.....	....	.....	44900		
....	....	...	.....	.....	.....	.....	.....	....	.....	44000	.....	oxygen removed
1018	acid	1+	few r and w	.....	.....	.....	.....	....	.....	26400		
....	....	...	.....	.....	.....	.....	.....	....	.....	.....	26400	
....	....	...	.....	.....	.....	.....	.....	....	.....	.....	26400	
....	....	...	.....	.....	.....	.....	.....	....	.....	.....	8800	
1020	alk.	2+	occ. wbc									
1020	acid	3+										
....	....	...	.....	.....	.....	.....	.....	....	.....	.....	.....	sitting in chair
....	....	...	.....	.....	.....	.....	.....	....	.....	.....	.....	walking

358,500 Oxford units of penicillin were given over the course of eleven days.

Within twenty-four hours following the inauguration of this therapeutic bombshell

status brightened, urinary and fecal incontinence became controlled, and pulse and respiration improved. The temperature was normal in forty-eight hours. Together with these

changes, too good to be true, hope for her recovery revived. Each blood culture drawn thirty-six hours, eighty-four hours, 132 hours and 180 hours after onset of treatment with penicillin was reported negative subsequent to forty-eight hours' incubation and also following a seven-day period of incubation.

The patient progressed steadily toward recovery. The temperature curve remained normal for the remainder of her hospital stay. On June 17th, she was permitted to dangle her legs over the edge of the bed; three days later she was allowed up in a wheel chair and on June 24th she was up and about anxiously awaiting discharge from the hospital which was granted on June 26th. The patient had been at the hospital fifty-seven days. Graphic representation of fever curve, therapy and laboratory findings are shown in Figures 1 and 2.

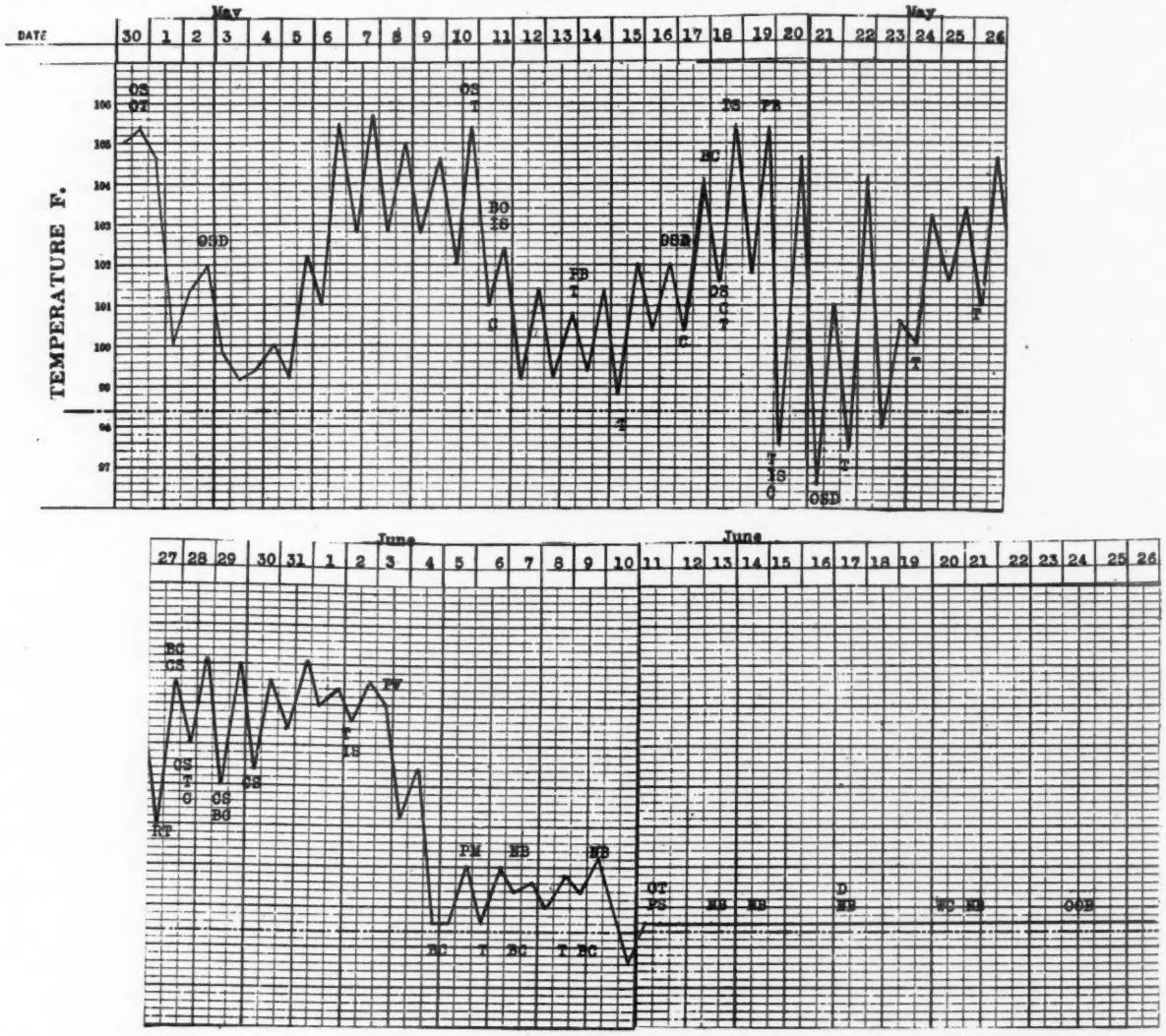

FIG. 2. T, transfusion of citrated whole blood; OS, oral sulfathiazole or sulfadiazine; OSD, oral sulfa drug discontinued; RT, rectal temperature; OT, oral temperature; C, chill; BC, blood culture taken; IS, intravenous sulfadiazine; PB, positive blood culture; NB, negative blood culture; CS, convalescent serum; PV, penicillin intravenously; PM, penicillin intramuscularly; WC, patient sitting in wheelchair; OOB, out of bed.

We wish to express our appreciation to Dr. T. S. Welton, Director of Obstetrics and Gynecology at Greenpoint Hospital, Brooklyn, N. Y. for granting permission to publish this case.

RUPTURE OF THE RECTOSIGMOID BY COMPRESSED AIR*

CASE REPORT

S. A. Swenson, Jr., M.D. AND Henry N. Harkins, M.D.
Assistant Resident Surgeon, Henry Ford Hospital — Associate Surgeon, Henry Ford Hospital
DETROIT, MICHIGAN

ALTHOUGH traumatic perforations of the rectum and the rectosigmoid are relatively well known, reports of cases of rupture by compressed air are limited in number. On the other hand, impalement on some object is a frequent cause of traumatic rectal perforation, and numerous cases have been reported.[2,5,6,7]

The condition of rupture by compressed air, which is a now well recognized industrial accident, was first reported by Stone in 1904.[9] Andrews, in 1911,[1] first brought critical attention to its pathogenesis, and pointed out that the jet of compressed air need only be pointed in the direction of the anus, the buttocks forming a funnel-like structure, directing the stream into the anus itself. In most reported cases the jet of air has passed through the patient's clothing which offers no protection. Burt,[4] in 1931, in experimental work showed that relatively small amounts of pressure were sufficient to cause perforation of the intestinal tract with variations according to the distance below the pylorus.

Brown and Dwinelle (1942)[3] recently reviewed the literature, finding sixty cases of complete perforation and nine cases of incomplete perforation caused by compressed air. These authors added two cases of complete perforation and one case of incomplete perforation, bringing the total to sixty-two and ten, respectively. In our review of the literature, we found one more case of complete perforation reported by Santy and Marion,[8] another reported by Ide (1942) and operated upon by Olson, and add one case of our own, bringing the total to sixty-five of the complete type. The longitudinal lacerations shown in an illustration in Ide's report are identical to those occurring in our own case. In both instances, incomplete lacerations involved only the outer layers of the large bowel.

Several factors in this case are of sufficient interest to warrant its report. First, the operation is an emergency procedure, and, due to variety in location of perforations, the method of repair is largely dependent upon the ingenuity of the operator. Preoperative preparation should be as extensive as possible even though time is an essential factor in morbidity and mortality. Sulfanilamide placed in the abdominal cavity is, by now, a well recognized adjunct, and should be used in any case of this type in which there has been contamination of the peritoneal cavity by intestinal contents.

CASE REPORT

B. A., an obese, white Italian male, aged forty-three, a laborer, was brought to the Henry Ford Hospital from an industrial plant on August 18, 1942. An hour before admission, while he was bending over a fellow worker turned a compressed air hose at the patient's buttocks and released a sharp blast of air. The patient fell to the floor and immediately felt sharp abdominal pain, and noted that he was "blown up like a balloon." The patient was taken to the First Aid room at the plant and was immediately transferred to the Henry Ford Hospital.

On admission he was seen to be well developed and well nourished, but in very evident and acute distress. History was obtained as above. Physical examination was essentially negative except for marked abdominal distention, rigidity and tenderness. The abdomen was highly tympanitic to percussion. Blood

* From the Division of General Surgery, Henry Ford Hospital, Detroit.

pressure was 165/115, pulse 88 per minute, and temperature 99°F. No external injuries were evident around the anus or peri-anal tissues. The blood Kolmer, Kahn, and Eagle tests were negative; the white blood count was 7,000 with 46 per cent polymorphonuclear neutrophiles and 54 per cent small lymphocytes.

A diagnosis of traumatic perforation of the rectosigmoid was made and immediate operation was decided upon. Continuous gastric suction was begun and the operation was done under spinal anesthesia, using 18 cc. of nupercaine. A midline incision was made from the pubis to half-way between the umbilicus and the xiphoid, exposing the peritoneum, which was seen to be bluish and stretched. When the peritoneum was opened, at least 2 liters (possibly 3 to 4 liters) of air gushed out under sufficient pressure to cause a whistling sound and resulting in a marked collapse of the distention. The bowel was explored from one end of the rectum to, and including the stomach, and four lacerations in the rectum and rectosigmoid were found. The remainder of the bowel showed considerable edema but no marked distention. There was some fibrin on all of the peritoneal surfaces associated with a small amount of free fluid but no definite evidence of fecal matter or peritonitis was found. Two complete longitudinal through-and-through lacerations of the rectosigmoid were sutured, the first being a linear one, was sutured longitudinally, while the second being an actual defect in the tissue, was sutured transversely. Both of these were about an inch long. Deep down near the rectum itself, were two one inch long longitudinal lacerations of the serosa alone; the mucosa pouched out but was not perforated. These were both sutured longitudinally in the direction of the tear as a precautionary measure. Silk was used as suture material throughout the operation. Six Gm. of sulfanilamide powder was inserted into the peritoneal cavity. The transverse colon was mobilized and brought through the omentum and out through the wound just above the umbilicus. The wound was closed without damage, using two layers of silk, heavy, for the fascia and peritoneum, and fine for the anterior layer after four heavy silver wire through-and-through retention sutures were placed. The patient left the operating room in good condition.

When the patient reached the floor he was immediately placed in an oxygen tent and continuous gastric suction was begun. A hypodermoclysis of 1,500 cc. normal saline and 500 cc. of 0.8 per cent sulfathiazole was also immediately instituted. The postoperative temperature was 102°F., and varied between 101° and 102°F. for four days when it receded to 98.6°F. and remained normal thereafter except for very slight elevations during the next few days. Hypodermoclysis of 0.8 per cent sodium sulfathiazole, 1,500 cc. were continued daily for four days after operation, then substituted by oral sulfathiazole, 6 Gm. every day for two days, as the gastric suction was discontinued on the fourth day. Fluid balance during the four days of continuous gastric suction was maintained by parenteral glucose 5 per cent and normal saline.

The extra-abdominal transverse colon was opened on the first postoperative day, forming a double-barreled loop colostomy. This colostomy opening functioned satisfactorily following irrigation with hypertonic saline and cotton-seed oil.

The patient's subsequent convalescence was essentially uneventful except for a delay in closing the colostomy due to technical difficulties not connected with the original accident. Interestingly enough, however, about three months following the accident the patient developed a small, superficial fistula-in-ano, and redundant hemorrhoidal tissue, both of which were removed at a subsequent operation.

SUMMARY AND CONCLUSIONS

1. Relatively few cases of traumatic rupture of rectum and rectosigmoid from compressed air have been reported in the literature.

2. We present one case of traumatic rupture (four lacerations: two complete and two incomplete) of the rectosigmoid caused by compressed air, with comparatively uneventful recovery. This case brings the total of cases with complete rupture reported in the literature to sixty-four.

3. Incomplete ruptures usually involve the serosa; the inner layers remaining intact. In most instances involving the rectosigmoid, the rupture is longitudinal whether complete or incomplete. Except

where there is an actual defect of tissue, such as occurred in one of the ruptures in our case, the tears may safely be closed in the direction of the tear, i.e., longitudinally.

4. Immediate operation is indicated when the condition is diagnosed or suspected.

5. Latent manifestations of anorectal disease might be considered as directly related to the initial trauma.

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Mere contact of bacilli of tuberculosis with the intestinal wall does not seem sufficient to produce intestinal tuberculosis. Factors favoring absorption of the organisms are trauma, diets deficient in vitamins, unusual virulence of the organisms and anaphylactic reactions of patients suffering from tuberculosis.

OSTEOCHONDRITIS DISSECANS*

CASE REPORT

THEODORE B. STRANGE, M.D.†
ALBANY, NEW YORK

OSTEOCHONDRITIS dissecans is defined as a localized aseptic necrosis of cartilage and underlying bone. It is usually found in adolescents and young adults and a great majority of the cases occur in the knee. A survey of the English literature for the past twenty years reveals no reported cases of this disorder in a patient as young as four and one-half years. Five cases of osteochondritis dissecans under the age of twelve years were reported, two in the hip and three in the knee. Freund[1] reported osteochondritis dissecans in the hip of a boy of six and one-half years. A plaster spica was applied and when it was removed three months later the patient showed no limitation of motion and no pain in the hip. This patient had had a Legg-Perthes's disease in the other hip for the previous year and a half. Gold[2] found this condition in the left hip of a boy eleven years. Conservative treatment led to improvement. Archer and Peterson[3] reported a case of osteochondritis dissecans occurring in the knee of a boy of ten years. Motion was markedly limited and while at operation the fragment could not be found, the patient regained perfect function in the knee. Fairbank[4] operated upon a case of osteochondritis dissecans in the knee of a girl of nine years. She had had

FIG. 1. The characteristic excavation of the cartilage and subchondral space of the femoral condyle is seen. The osteochondritic mass is almost completely detached.

FIG. 2. The semi-detached mass has disappeared and the cavity has become obliterated.

* From the Department of Orthopedic Surgery, Albany Hospital. † Dr. Strange is now a Lieutenant in the Medical Corps of the United States Naval Reserve.

pain and swelling for the previous six months. The results of the treatment were not noted. Freund[1] also states that he heard of the case of a boy of ten years, who was said to have had a "free joint body" removed from his knee joint.

This case is reported because it is a child of only four and one-half years. The youngest child with osteochondritis of the knee reported in the literature reviewed was nine years. It is thus seen that while it is thought that this is a disease of adolescence, this is not necessarily true. It is also worthy of note that in this case, conservative treatment led to complete cure, whereas open operation is usually recommended.

CASE REPORT

A boy of four and one-half years was admitted to the Pediatric Service of the Albany Hospital on October 21, 1941. His chief complaint was general malaise and moderate lameness in both legs for the previous three days. The mother had also noted a slight limp in the left leg for that period. Physical examination was negative except there was slight limitation of motion of the left knee and some pain was caused by this motion. Blood findings, including calcium, phosphorus and phosphatase were normal. Examination of the spinal fluid revealed no abnormalities. Anterior poliomyelitis was suspected as this boy was admitted in the midst of a severe epidemic of that disease. However, this was quickly ruled out.

X-ray of the left knee on October 22, 1941, (Fig. 1), revealed a definite small defect, apparently in the medial posterior articular surface of the epiphysis of the femur. Within this defect, but almost completely detached, was a small fragment of bone measuring about 2 mm. in dimension. Diagnosis of osteochondritis dissecans was made and the patient was transferred to the Orthopedic Service and a plaster cast applied to the extremity with the knee in a slightly flexed position. The patient was discharged from the hospital, but was not allowed to bear weight on the leg. On December 6, 1941, the cast was removed, after having been in place for six weeks. *X*-rays at this time showed that the defect had filled in completely. The child was allowed to resume weight bearing and *x*-ray on February 4, 1942, (Fig. 2), showed that the defect had remained healed. At this time the patient had absolutely no symptoms, examination was entirely negative and the patient was regarded as entirely well three and one-half months after the onset of his symptoms.

SUMMARY

A case of osteochondritis dissecans occurring in the knee of a child of four and one-half years is reported. Conservative treatment led to complete cure within a few months. It is thought that this is worthy of report. As far as can be noted, no case has been reported in one so young.

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MESENTERIC THROMBOSIS OF THE TERMINAL ILEUM*

CASE REPORT

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Consulting Surgeon, Bellevue Hospital

AND

WILLIAM T. DORAN, JR., M.D.
Assistant Visiting Surgeon, Belleveue Hospital

NEW YORK, NEW YORK

THERE have been seventeen cases of mesenteric thrombosis in Bellevue Hospital during the past five years. One patient left the hospital one hour after admission and before operation; fifteen patients have died and this is the report of the seventeenth case.

This case report is presented to illustrate one thing namely, that the McBurney incision was most adequate in this situation. The resection of the lower ileum and secondary anastomosis to the ascending colon was accomplished without difficulty and without waste of time or shock to this patient and with no sacrifice of maximum utility to the patient.

CASE REPORT

A seventy-seven year old white male was admitted to the Fourth Medical Division of Bellevue Hospital on August 26, 1941, at 3:00 A.M. On admission he stated that he had been attending clinic for one month for arthritis which had somewhat restricted his activity. Despite this he had noticed some shortness of breath on exertion during this period. One hour before admission he awoke with pain all over his abdomen which caused him to vomit food but no blood. The pain rapidly increased in severity but had remained generalized, although it became more marked in the upper abdomen. For fifteen minutes before admission the patient had a chill and complained of feeling weak, cold and clammy.

His past history revealed that he had undergone a double inguinal herniorraphy in 1918. There was no cardiac or intestinal symptomatology except moderate chronic constipation. Venereal disease was denied.

Physical examination revealed an acutely ill, elderly, small moderately well nourished white male. His temperature was 98°F.; pulse 4,8 regular, blood pressure 180/95. The abdomen was not distended but was tender throughout; this tenderness was most marked in the lower abdomen. No rigidity or involuntary spasm was elicited. There was rebound tenderness throughout. The cardiac examination was not remarkable; urinalysis was negative; white blood count 21,000; polymorphonuclears 92 per cent.

After admission the pain subsided somewhat. The patient appeared subjectively improved; tenderness became more localized to the right lower quadrant with moderate spasms. His blood pressure fell to 130/85; bradycardia persisted below 60. The patient vomited twice after admission and had a normal bowel movement shortly after admission both of which were negative for blood. The Wassermann test was negative.

Surgical consultation was asked and it was our opinion that acute appendicitis was the most probable diagnosis. Celiotomy was performed thirteen hours after admission after supportive infusion was given.

Cyclopropane anesthesia was used throughout. A standard McBurney incision was used. When the peritoneum was opened sanguineous exudate was found in the abdomen. A gangrenous intestine presented moderately distended. On investigation this proved to be ileum and to involve the lower four feet of ileum to the ileocecal valve. The mesentery showed thrombosis of the branches of the superior mesenteric artery and its radicals in a fan-shape supplying this area. The ilium was resected and an end-to-side iliocolostomy performed to the ascending colon at a point approximately two inches distal to the ileocecal valve. An ileostomy was performed one

* Presented before the Section on Surgery of the New York Academy of Medicine, April 10, 1942.

foot proximal to the site of anastomosis, and 10 Gm. of sulfanilamide powder were placed in the abdomen.

Sulfathiazole, transfusions, oxygen tent, infusions and Miller Abbott suction were used postoperatively. Fluids were allowed shortly after operation. The ileostomy tube was removed on September 1st, the sixth postoperative day. Sutures were removed on the ninth day after operation. The ileostomy fistula closed spontaneously on the eighteenth postoperative day and the patient was discharged on September 22nd, the twenty-seventh day after operation.

The pathological report showed the gangrenous bowel lumen to contain exudate and transudation of red cells and typical infarction of the mesenteric vessels.

An inflammatory condition, similar to regional ulcerative colitis, may involve segments of the small intestine, usually the terminal part of the ileum. Pathologically, this condition is somewhat like "chronic ulcerative colitis." The history will usually be of long standing, recurrent, progressive diarrhea, with abdominal cramps, loss of weight, low grade fever and anemia.

The brief excerpts in this issue have been taken from "The Modern Management of Colitis" by J. Arnold Bargen (Charles C. Thomas).

New Instruments

A NEW RHINOMETER

MORTON I. BERSON, M.D.
Director of Plastic and Reconstructive Surgery, The Downtown Hospital and Pan-American Clinic
NEW YORK, NEW YORK

TO obtain esthetic results in rhinoplasty, the author has devised a sterilizable rhinometer. It is useful in the diagnosis of nasal deformities and is valuable in progressive checking of the required normal measurements during nasal operations.

The instrument consists of the following parts:

A. *Two rests* fitted under the superior orbital crests in conjunction with the base of the platform D, which is placed at the columella lip junction, act as fixed points from which accurate measurements are made.

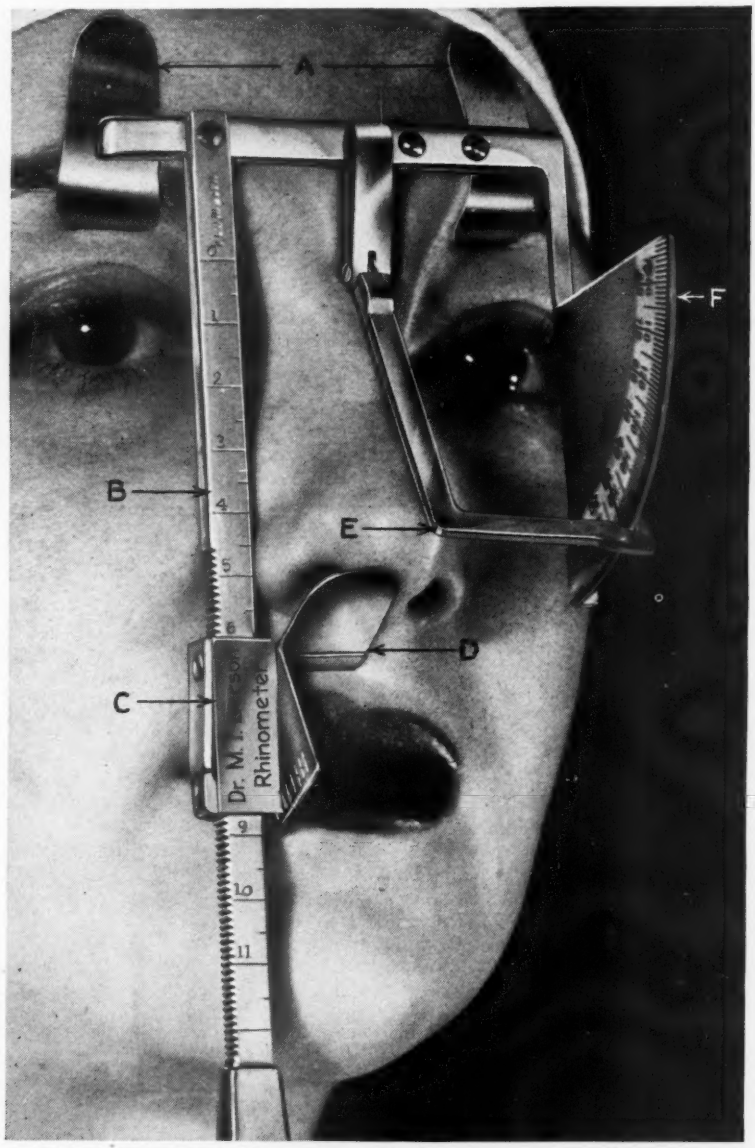

FIG. 1. Author's rhinometer. (*The Laryngoscope*, April 1943.)

B. *A vertical column with a numerical scale* designed to carry the sliding platform D, and to record the length of the nose.

C. *A cross-member carriage* designed to slide the platform to various levels along the column. Then according to the graduated scale, the difference between the actual length (from nasion to apex) and the desired length is measured.

D. *The platform* fits under the columella lip junction of the nose and can be tilted to the desired esthetic angle registered on the calibrated scale.

E. *The lever on the calibrated arc* F is adjusted to the normal profile angle, i.e., the degree of projection of the nose from the face, and the excess to be removed is then determined.

By the use of the rhinometer, the following absolute measurements can invariably be attained. *The length* of the ideal nose is one-third of the facial length. The superior third is from the hairline to the nasion. The third occupied by the nose is from the nasion to the tip of the nose. The lowest third is from this point to the tip of the chin or gnathion. *The normal tilt angle* is 95 degrees; this varies slightly according to the relationship with the lower third of the face. In cases of short or receded chins, this angle can be tilted to 105 degrees. *The profile angle* or projection of the nose is determined by a line drawn from the glabella to the root of the nose. The lever E is so adjusted to measure the desired height of the nose on the calibrated scale F. The ideal nasal profile angle should measure 30 degrees.

The rhinometer is important in determining the exact amount of tissue to be removed, and besides its practical value, it has also a reassuring effect on the patient when he or she sees that the surgeon is not operating haphazardly but has a definite scale and plan.

SURGEON'S THUMB GOWN

A. E. Drexel, M.D.
Attending Surgeon, Halifax District Hospital
DAYTONA BEACH, FLORIDA

I doubt if many surgeons can put on a pair of sterile dry gloves without at some time during the procedure directly or indirectly cause some portion considered as unsterile to come into momentary contact with the sterile exterior of the glove. This fact can be proved by making the following experiment: Paint unsterile parts with some highly colored solution and then see where it may be deposited when the gloves are on. The so-called *thumb gown* makes this error practically impossible.

Fig. 1.

An ordinary surgeon's gown with a stockinette cuff is required. In the sewing room, maintained in almost all hospitals, a strip of ribbon tape is sewed to the end of the cuff into a loop as illustrated in Figure 1. The thumbs find these loops without any difficulty when putting on the gown. A nurse holds open the gloves and with one diving motion the surgeon's hands are placed full depth into the gloves without further ado. The surgeon never touches either glove with the other hand.

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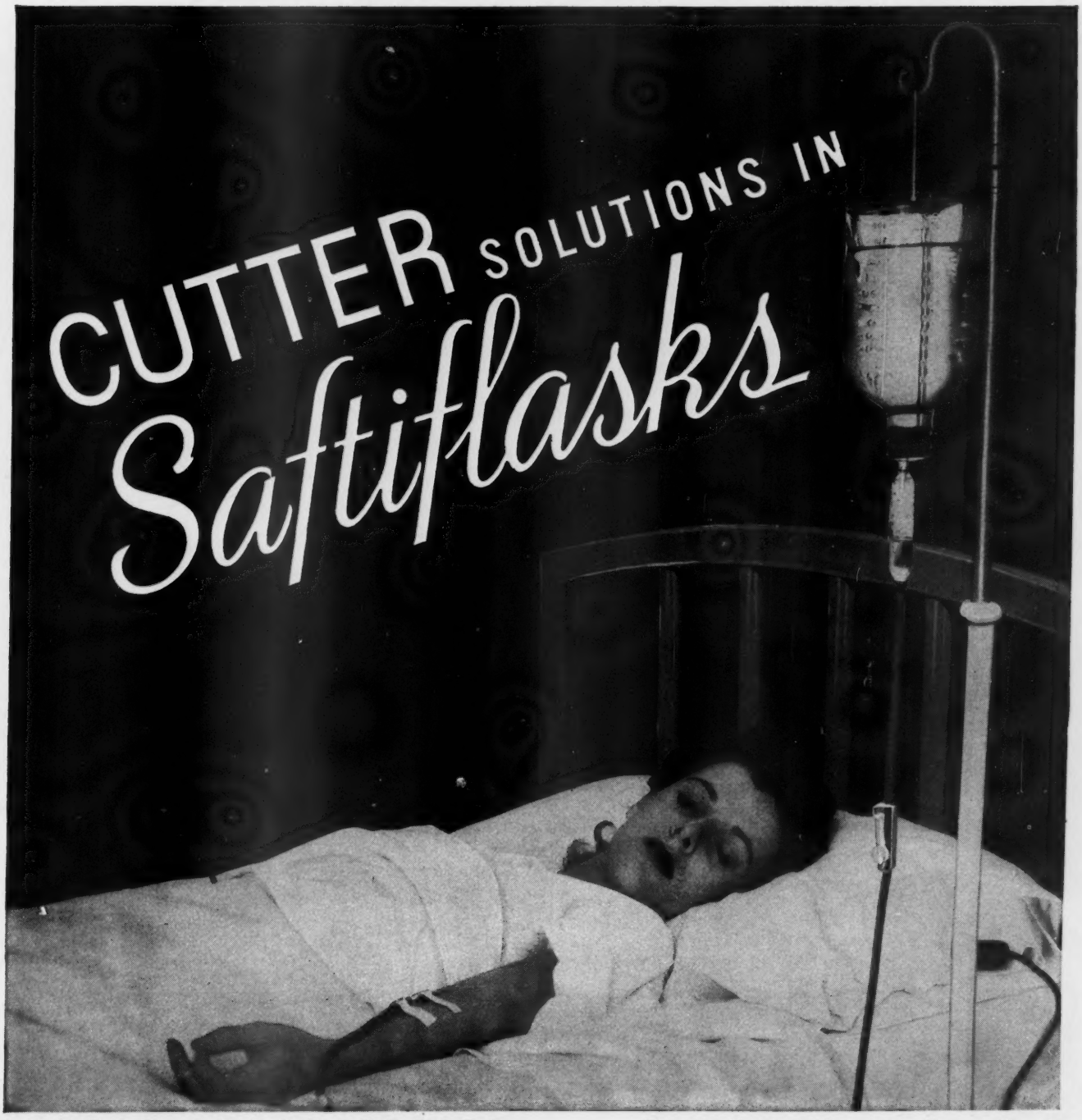

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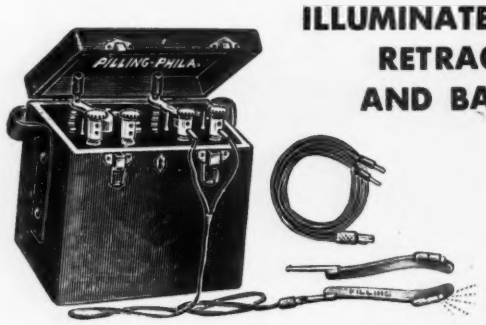

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P15185—Battery, Frazier, two-circuit for brain retractor, provides two absolutely independent circuits, is an assurance against current supply failure during operation (includes dry cells, but does not include cords or retractors, suitable for Set No. P15160 shown on right.

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P15156—Lamp for Brain Retraction, clear.

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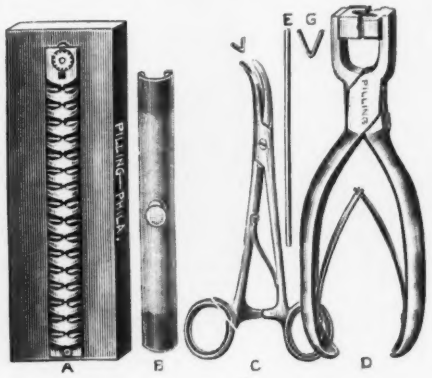

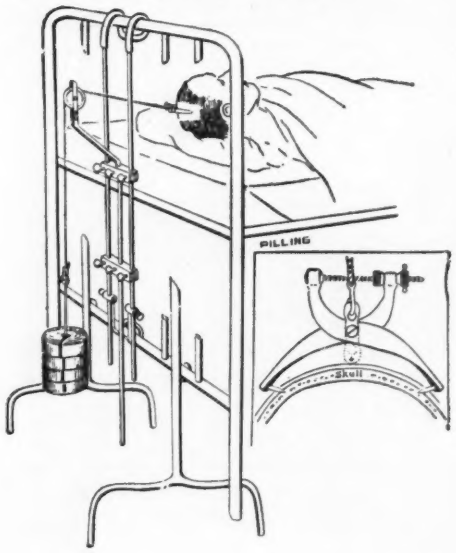

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W. Gayle Crutchfield, M.D., Dept. of Neurological Surgery, Medical College of Virginia, Richmond, Virginia.

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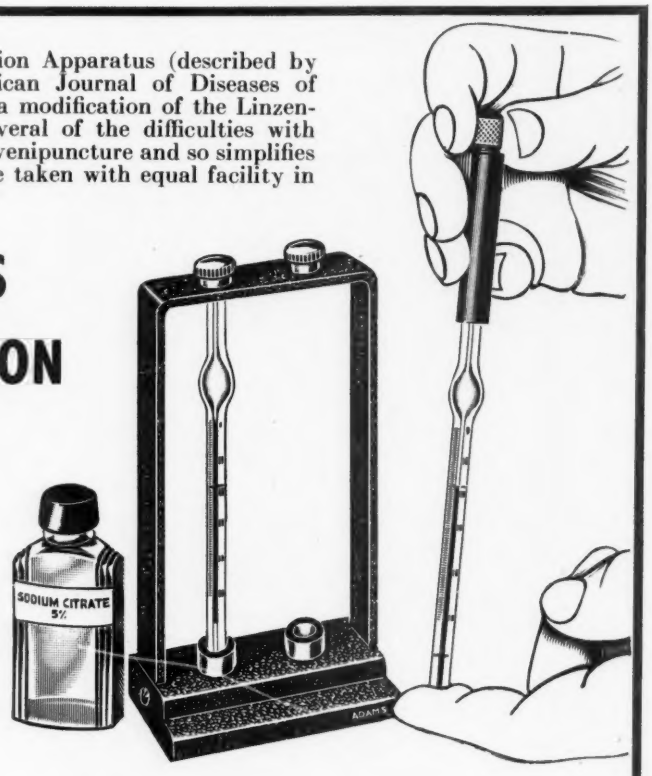

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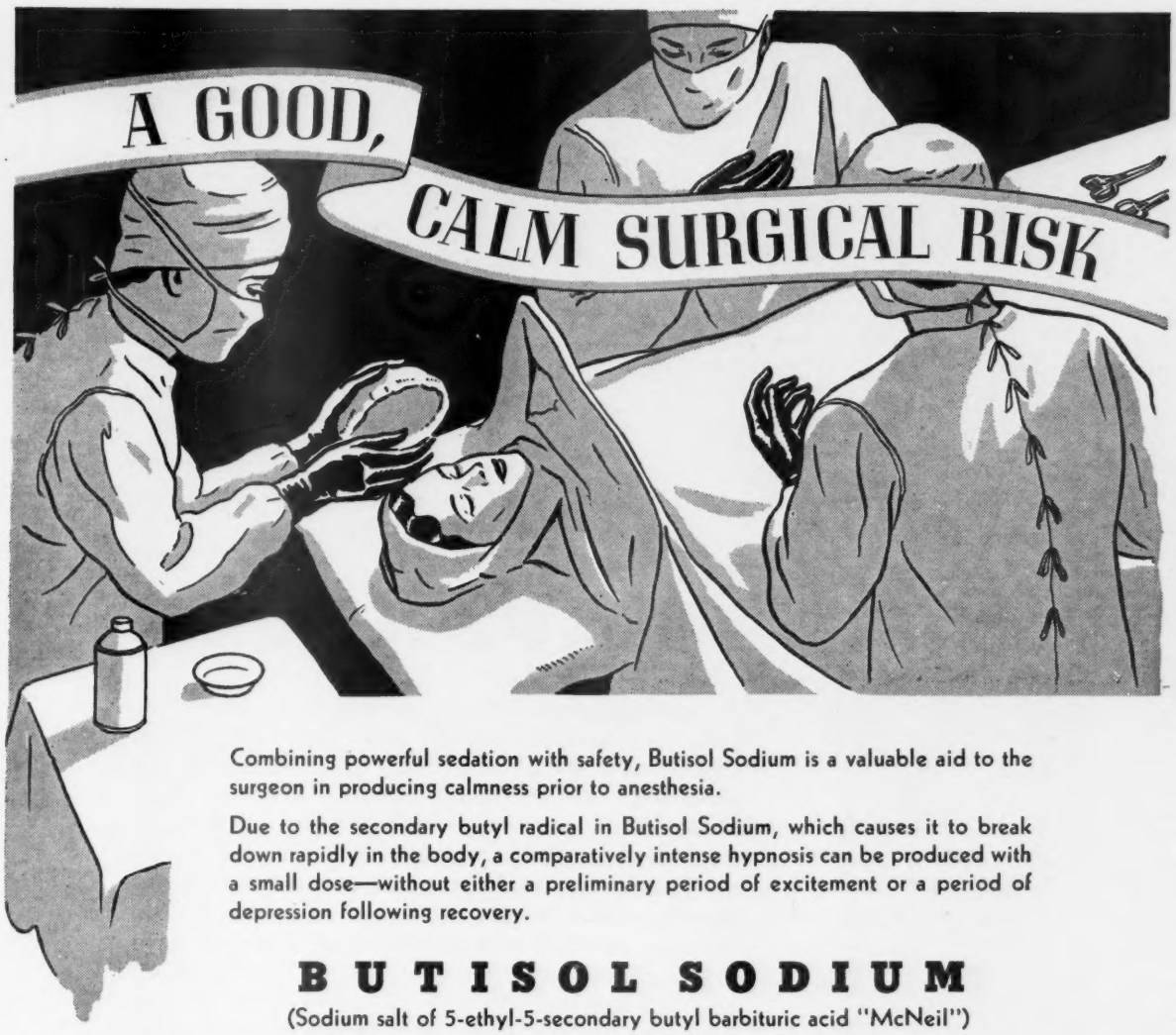

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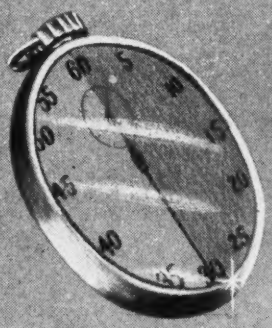

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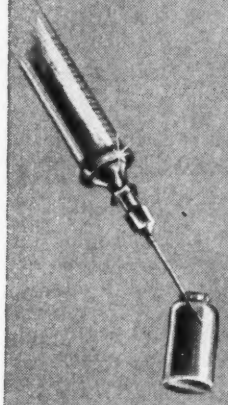

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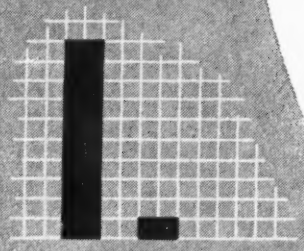

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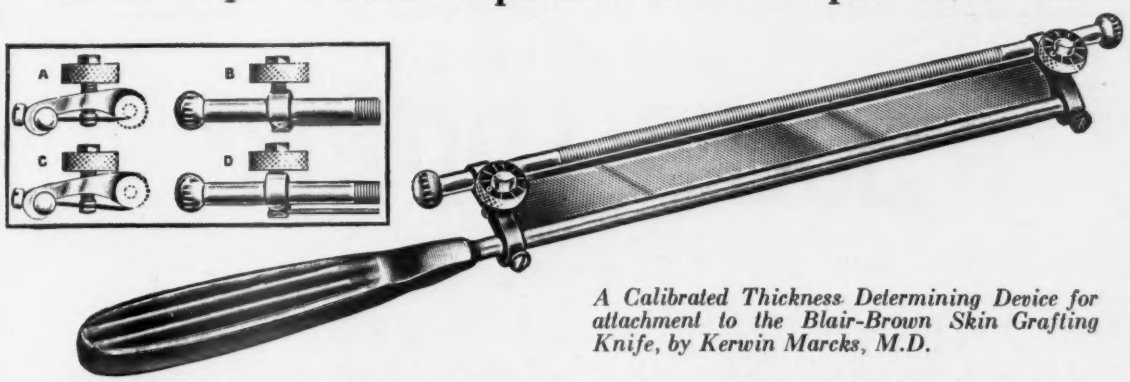

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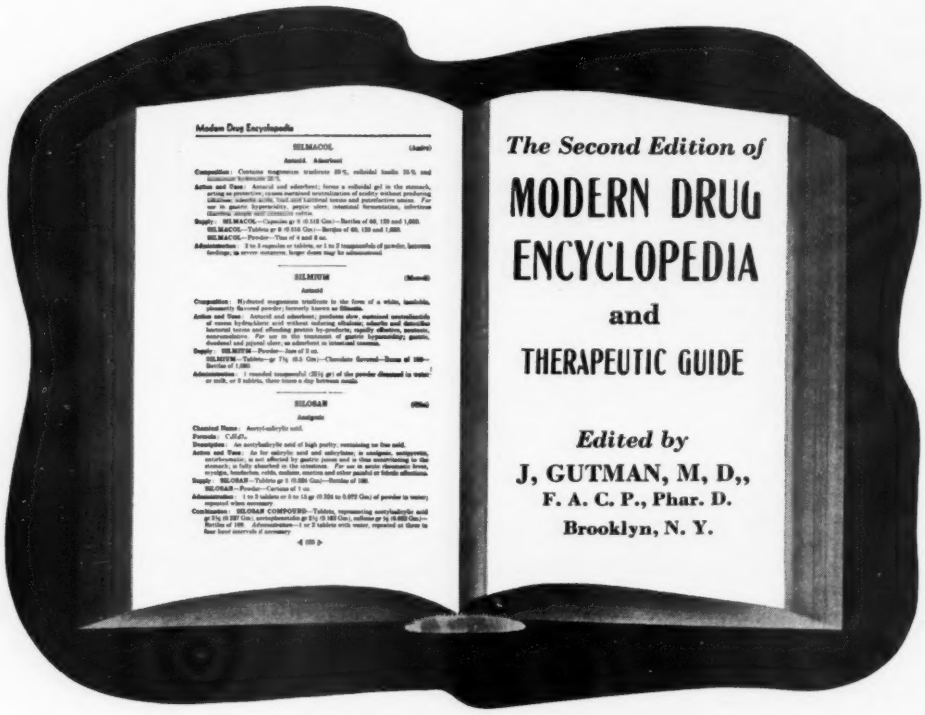

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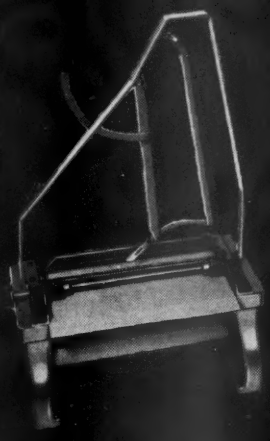

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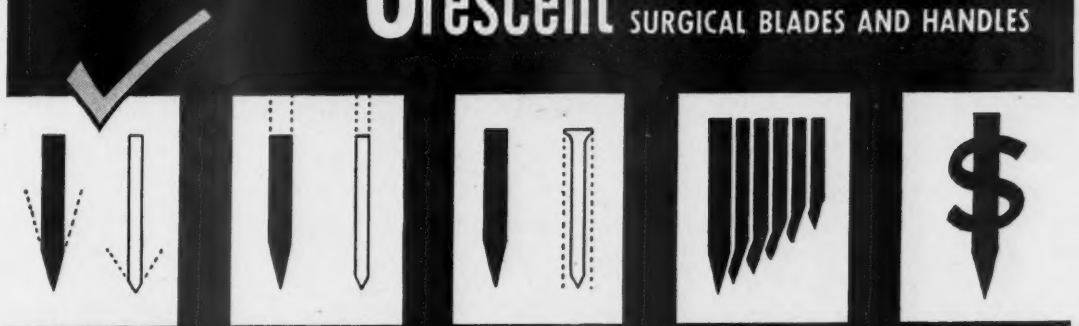

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[1] Clement, F.; Elder, C.: Anesthesiology, 4:516, 1943.

[2] Forsee, J.; Shefts, L.; Burbank, B.; Fitzpatrick, L.; Burford, T.: J. Lab. & Clin. Med., 28:418, 1943.

*Trade Mark Reg. U. S. Pat. Off. Word "Nupercaine" identifies the product as alpha-butyloxycinchoninic acid - gamma - diethylethylenediamide-hydrochloride.

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